POPULAR SCIENCE

Stroke and Rehabilitation

M.C. Maheshwari
K.K. Singh

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Preface

Stroke is the third most important cause, after cancer and heart attack, of morbidity and mortality in the elderly population of any nation. Stroke can occur at any age, but is more frequent in past middle-aged men and women. There have been significant and useful advances in the understanding of the causes, diagnosis, and management of stroke in the last few decades. These developments have led to the establishment of a new clinical service through 'stroke units'. Stroke units are keeping pace with the patient's expectations in quick and effective health care delivery. These stroke units have justified the need for the stroke patients to be treated at the same speed as heart attacks in coronary units. New concepts and therapeutic methods have changed the outcome of stroke patients. These in turn have resulted in significant changes in medical and public attitudes towards stroke.

Hardening of the arteries with blockage of a blood vessel with a clot is the commonest cause of stroke. Several risk factors—preventable and non-preventable—have been identified. Passing, brief attacks of ischaemia which may manifest as transient blindness, weakness of arm or leg or fainting are very important warning signals and should not be neglected. Recognition and effective control of high blood pressure in the society has definitely lowered the incidence of stroke and its morbidity and mortality. There is an acute need for creating awareness about the benefits of preventable risk factors. The best time for prevention is at school or college age. Regular exercise and a well-balanced

nutritious diet should be made an integral part of one's lifestyle. Smoking and alcohol should be avoided. High blood pressure and diabetes mellitus should be diagnosed at the earliest and necessary treatment initiated.

Stroke is one of the important causes of severe disability in the society. Stroke patients, as against heart attack patients, rarely return to their jobs and social positions. The aim of the rehabilitation is to determine realistic goals to obtain optimal functioning despite some disability. Rehabilitation deals with the development of a person to the fullest physical, psychological, social, vocational, and educational potential consistent with his or her physiological or anatomical impairment and environmental limitations. Rehabilitation measures should be started as soon as possible after the occurrence of a stroke and need to be continued until objectives are achieved.

This book is primarily written for the lay people, patients of stroke and their relatives, and for the general medical practitioner. The brunt of medical care in later stages falls mainly on the family physician. The chapters have been divided under separate headings for better understanding and appreciation. Difficult medical terminology has been reduced as much as possible. Importance has been given to practical aspects of rehabilitation through exercises presented in simplified illustrations and pictures to enable the patients to do them at home.

We are very grateful to our families for showing patience and for providing the right milieu and encouragement to complete the task in time.

We wish to express *our* gratitude to Dr V. Singh, Dr S. Wadhwa and Dr S.L. Yadav of the Department of Rehabilitation and the staff for their continued cooperation which made the task of writing a pleasure.

We are extremely grateful to the National Book Trust, India for bringing out this book, and for providing continuous help, encouragement and support.

It is impossible to write on any subject without

expressing a feeling of gratitude to all those patients who have directly or indirectly contributed to our knowledge.

We would like to express our thanks to Mr H.K. Sharma, senior artist in the Department of Anatomy for drawing the CMET sections, and to the Department of Neuroradiology for CT and other investigations. Drawings in the chapter on rehabilitation were loaned by the WHO but were redrawn at NBT. We also acknowledge the secretarial assistance of Mr R. Sharma, Miss Anjana, Mrs C.K. Khurana of the Neurology Department and the staff of the Department of Rehabilitation for their help.

M.C. MAHESHWARI
K.K. SINGH

PART I

STROKE

M.C. Maheshwari

1

INTRODUCTION

The term 'stroke' is used to denote neurological dysfunction of the brain due to diseases of the blood vessels or the vascular mechanism. The World Health Organisation (WHO) has defined stroke as rapidly developing clinical signs for localised, and at times more widespread, disturbance of the cerebral function lasting more than twenty-four hours or leading to death with no apparent cause other than that arising from dysfunction of the blood vessels. The other commonly used terms for stroke are *phaliz, adhrang, lakwa,* or paralysis. Cerebrovascular diseases or strokes are very common neurological disorders and every physician either at primary, secondary or tertiary health care level is required to render medical help to these patients. Every general physician needs to know something about strokes and should have the competence to recognise them and refer to specialists where there are reasonable prospects of successful medical treatment and facilities for surgical intervention. Stroke is the third most important cause after cancer and heart attack of morbidity and mortality in elderly population of a nation. Until a few decades ago, attitude of the medical fraternity and the government and other funding agencies toward strokes was quite negative. Accordingly there was disproportionate expenditure and expansion of services with relation to heart care. This was so as the research developments and understanding of cerebrovascular diseases (diseases of the blood vessels supplying the brain) lagged behind the therapeutic goals. All this has changed now. Modern researches in the

understanding of such diseases have led to new methods of treatment which have very clearly changed the prospects of survival of stroke patients. Both mortality and morbidity have been reduced. These have resulted in significant changes in medical and public attitudes towards stroke. There is a need for continuous campaign to focus medical and public attention to the fact that a stroke victim, like the heart attack victim, needs urgent intensive care. There is very rightly an increasing budgetary demand to establish stroke units or centres with intensive care facilities for patients and for research purposes.

Story of the various factors that lead to cerebrovascular diseases has established beyond doubt that the best course to tackle the problem is primary prevention. Brief, passing ischaemic attacks are very important warning signals. Every transient ischaemic attack results in some damage to the nerves even if there are no clinical signs of any disturbance after twenty-four hours or earlier. It has been found that within five years of a passing attack, 35 per cent of the patients suffer a full-blown stroke. Even more significantly 50 per cent of these have their stroke within one month of their first attack. Therefore secondary prevention by treatment with drugs that reduce blood-clotting platelet formation or surgery is of immense value in order to avoid a full-blown stroke later.

Early diagnosis and management of acute stroke is as important and essential as of acute heart attack. There is plenty of evidence which shows that in the first few hours after stroke, part of the brain tissue is still viable and may be salvaged, thus improving the outcome for the patient. Advances in imaging techniques by CT scan or magnetic resonance imaging (MRI) have greatly benefited the management of stroke. The minute anatomical details provided by MRI convey precise information about the network of blood vessels in the brain. During the first few hours after the event, there are continuous changes in the brain tissue around the 'damaged' area. These are changes in blood

flow metabolism, amino acid concentration and other parameters. These are studied by new techniques such as single photon emission computerised tomography (SPECT) and positron emission tomography (PET) which have in turn contributed significantly to our understanding of what happens during a stroke.

Stroke is the commonest cause of severe disability in the aging population of any country. It produces much more devastating effects and the fortunate survivors, even if they suffer only limited impairment of the nervous system, rarely return to their jobs and social positions. Patients who survive a heart attack on the other hand normally return to their former jobs and social positions. This has led to poor lobbying for stroke patients to safeguard themselves and resulted in a nihilistic approach by doctors and the society. Recent understanding of the extraordinary capacities of the brain to compensate for damages suffered in a stroke should change this attitude. Though there is some specialisation or localisation of functions in the brain which are distributed over the larger area of the brain, there is a possibility of functional replacement. Other areas of the brain take over the responsibility of functions that have failed as a result of a stroke. All efforts should be made to rehabilitate the stroke survivors in their previous jobs and society. Doctors at the primary health care level should not feel reluctant to send stroke patients to specialised hospitals. There should be an ongoing continuous cooperative effort between neurologists and rehabilitation medicine. The rehabilitation medicine department should not only provide physiotherapy but all rehabilitative facilities which stroke victims require.

These include speech therapy and occupational therapy. Rehabilitation should start at the earliest possible moment because the opportunities lost at that early stage can never be recovered later on.

2

ANATOMY OF THE BRAIN

The brain develops from a structure called the ectodermal neural tube in the foetus. By the end of the fourth week of gestation, the neural tube is completely formed and closed. After closure there is a narrow tail-like structure which develops into the spinal cord. The enlarged front part of the structure develops into the brain. It grows into the three primary divisions of the brain: the hind brain, midbrain, and forebrain. With continued growth and development of the brain, individual structures get differentiated.

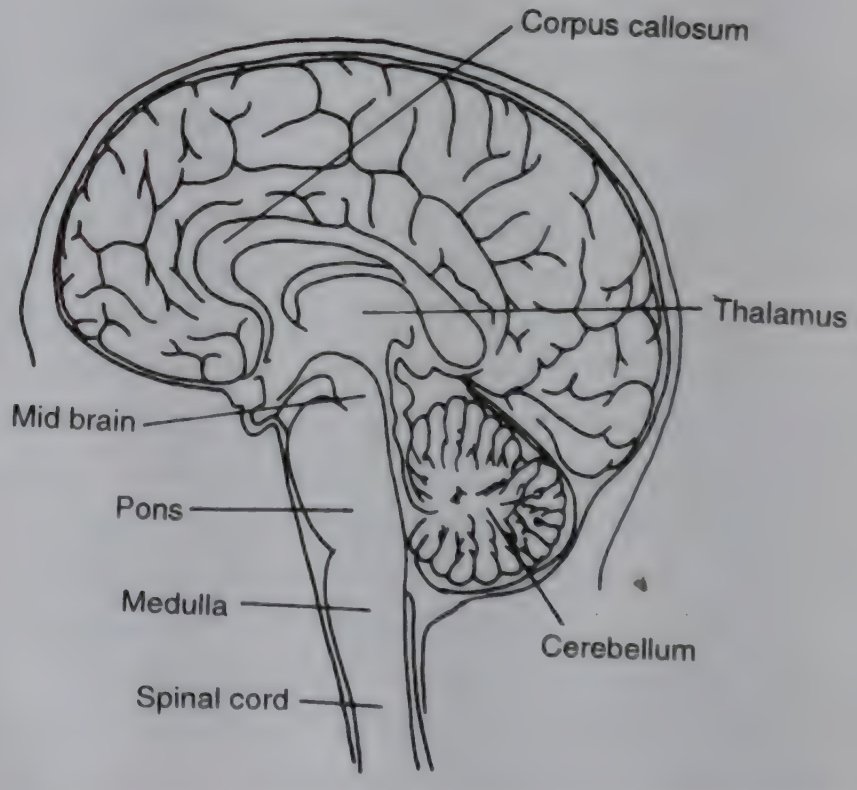

Fig. 1 : The adult brain with the corpus callosum which connects the two hemispheres.

The adult brain consists of two cerebral hemispheres which are connected by the corpus callosum (Fig.1). The surface of the cerebral hemisphere resembles a walnut with its convoluted surface. Each hemisphere is divided into frontal, parietal, temporal and occipital lobes (Fig.2). On the

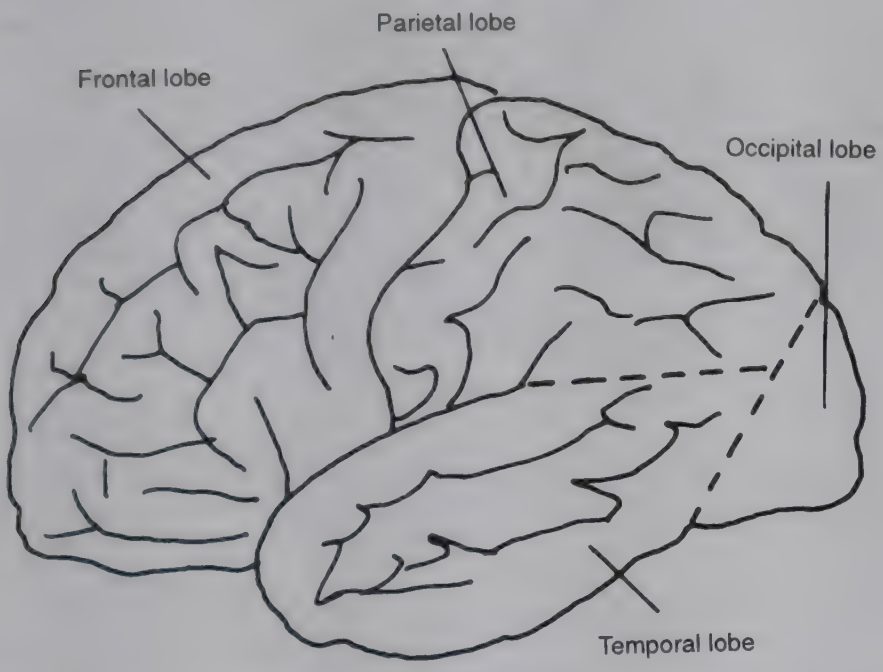

Fig. 2 : The external surface of the brain hemisphere with the demarcations of frontal, parietal, temporal and occipital lobes.

surface, the lobes are subdivided further into areas on the basis of function, i.e. motor, sensory, speech, visual, hearing, etc. (Figs. 3, 4). Immediately below the cortex is the corpus callosum. Below this are the basal ganglion, thalamus, hypothalamus (pituitary gland), midbrain, and pineal body. Below the midbrain are the pons, medulla oblongata and cerebellum (Fig.5). Like the cerebrum, the cerebellum has a cortex with folds. The cerebellum has two hemispheres united by a median vermis. Medulla oblongata continues as the spinal cord. The skull cavity which contains the brain, is divided into three fossae (depressions) by tough fibrous partitions into anterior (front), middle and posterior (rear) cranial fossa (Fig.6). Cerebellum and the greater part of the brain stem occupy the posterior cranial

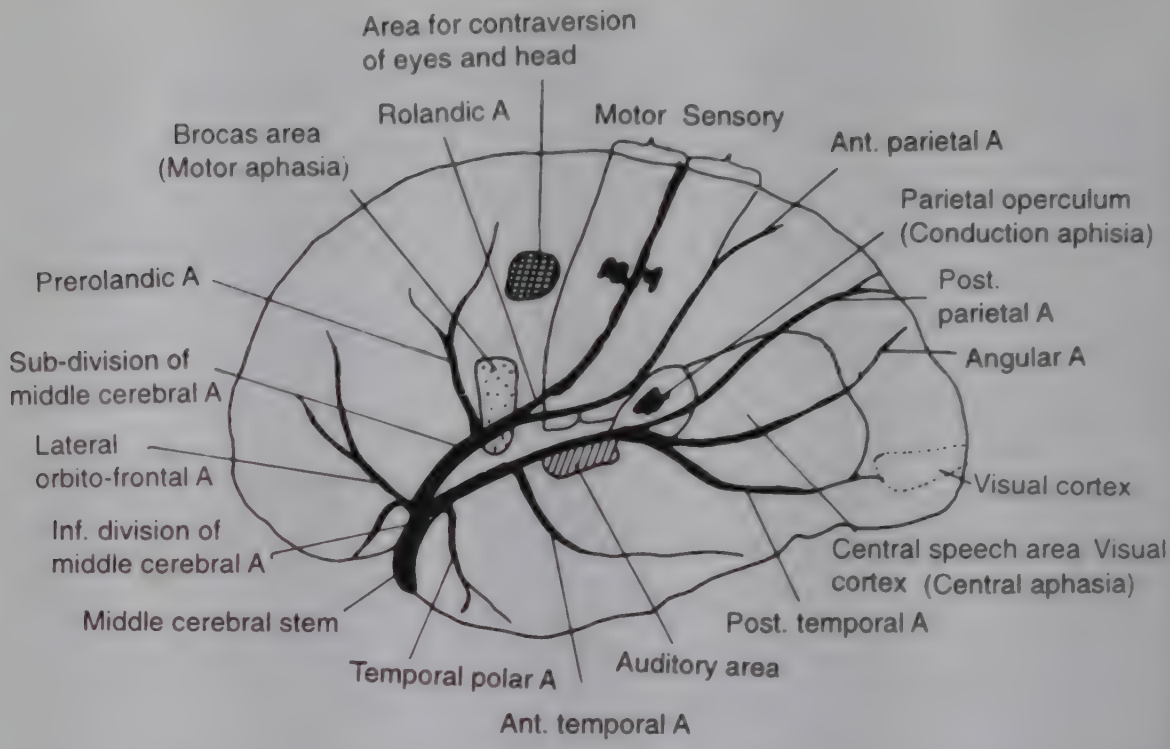

Fig. 3 : The external surface of the hemisphere with the functional areas and blood supply.

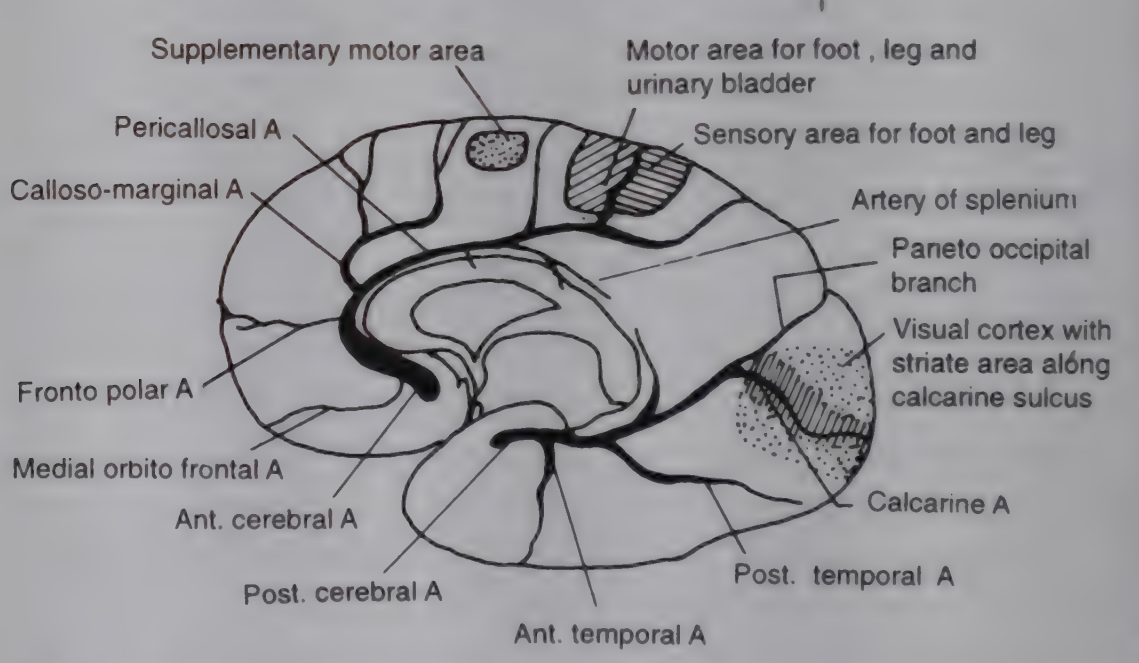

Fig. 4 : The medical surface of the hemisphere with representation of some functional areas and blood supply.

fossa. Temporal lobe occupies the middle fossa. The greater part of the anterior fossa is occupied by two cerebral hemispheres. The vertebral column or the backbone

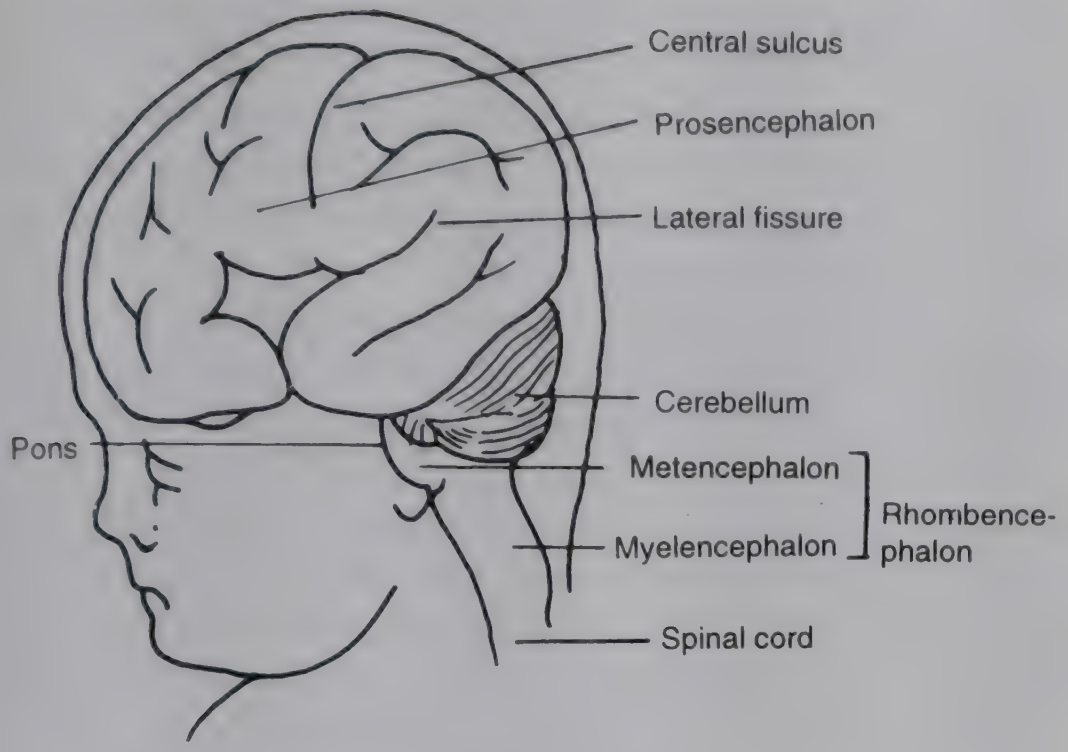

Fig. 5 : The various parts of the brain during early development.

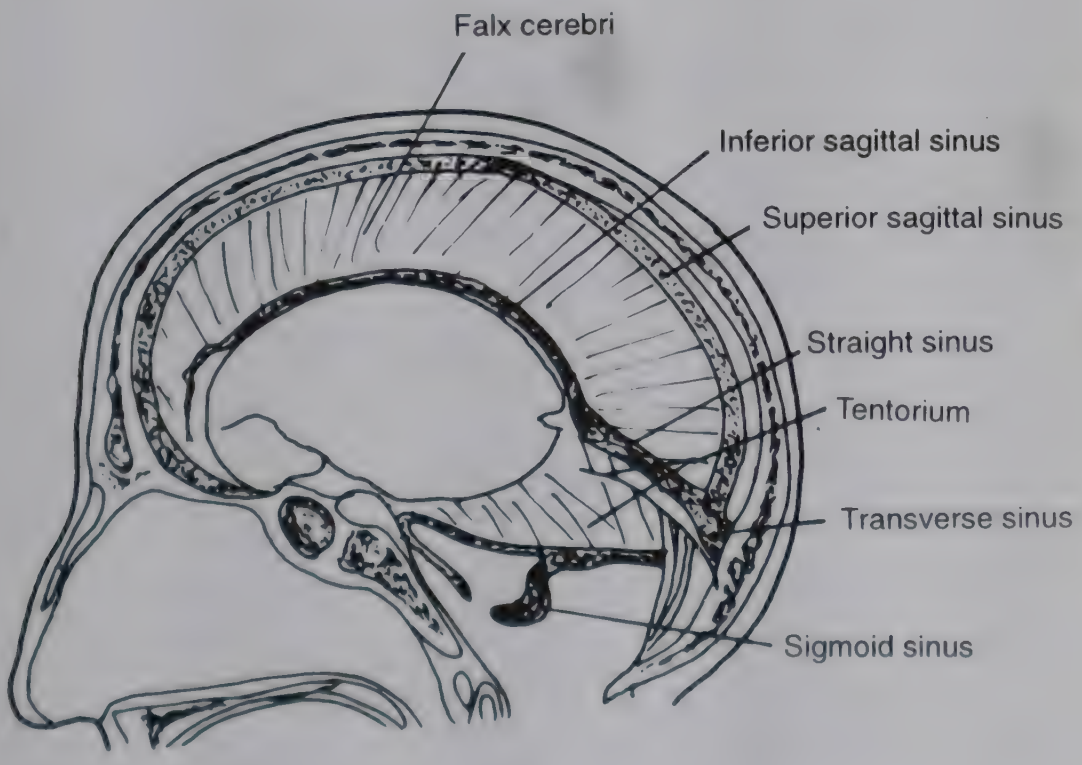

Fig. 6 : The tough fibrous layer—falx cerebri separating two hemispheres, and tentorium separating two hemispheres and the cerebellum. Some venous sinuses are also shown.

encloses the spinal cord. The brain and the spinal cord are enveloped by a thin continuous membrane called the meninges. The outermost meningeal layer is called dura mater and is the toughest. It almost sticks to the skull bone. The entire brain and the spinal cord is bathed in a watery fluid called cerebrospinal fluid.

All body functions are represented in the brain, some in the left and some in the right hemisphere. For many years neuroanatomists, neurophysiologists and neuropsychologists talked about the dominance of a hemisphere or its specialisation and localisation. For example, language is represented in the left hemisphere. Musical appreciation and construction ability is represented in the right hemisphere. Recent observations on stroke patients, especially

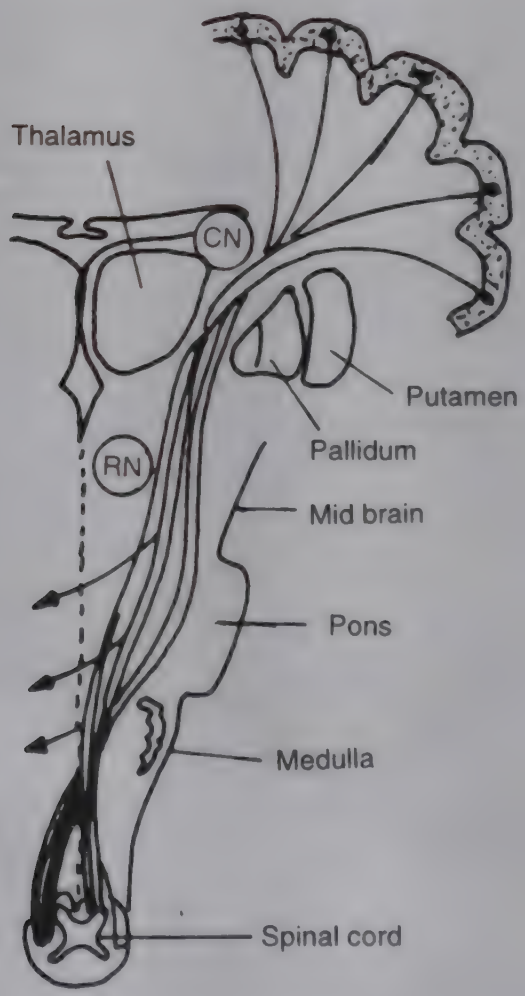

Fig. 7 : The course of pyramidal (motor) fibres in the neuraxis.

their ability to regain certain faculties lost after a stroke, however, cast doubt over such rigid localisation of the brain. Some experts believe that neurological functions are distributed over the entire brain even though some areas are more important than others. There is, therefore, a possibility of function replacement during rehabilitation after a stroke. The areas of the brain which usually remain dormant assume the responsibility for the functions when commonly recognised areas have failed to function because of a disease or a stroke.

It is very important for the readers to remember that right-sided functions like motor, sensory or visual fields have representation in the left hemisphere and left-sided functions are represented in the right hemisphere. The nerve fibres, both motor and sensory, carrying messages from and to the brain, cross at different levels in the central nervous system (Fig. 7, 8). The knowledge of the site where

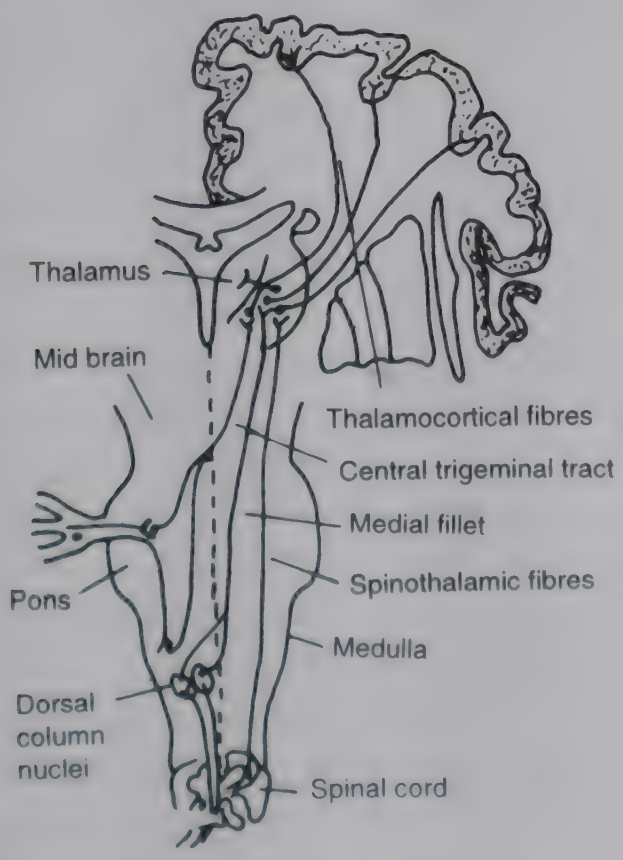

Fig. 8 : The course of sensory fibres in the neuraxis.

the fibres cross is important in localisation of the damaged area. Cerebellar cortex and its connections control the body functions on the same side, i.e. their action is ipsilateral. Similarly, cranial nerves which originate in the brain, have ipsilateral functional control. Some functions like consciousness, sleep, etc. have bilateral representation. This functional knowledge is important in localisation of the damaged area of the brain at the bedside.

The heart is an important organ as it continuously pumps the oxygenated blood and perfuses all the tissues of the body through the network of arteries (Fig.9). The

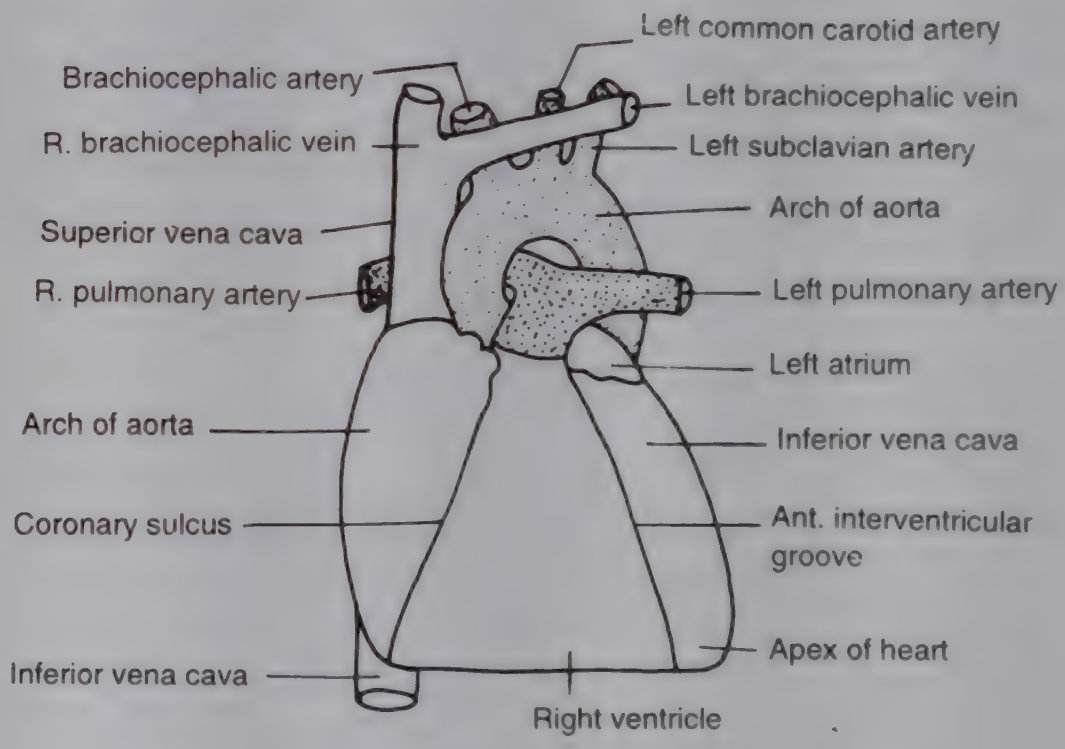

Fig. 9 : The heart and its major arteries and veins.

heart receives the purified or oxygenated blood from the lungs. The brain receives blood through two systems, called carotid, and vertebral-basilar system. As blood flowing through the carotid system does not normally mix with the vertebral-basilar blood, these two separate systems were recognised a long time ago. From the heart the blood flows into the aorta. The aortic arch gives rise to three

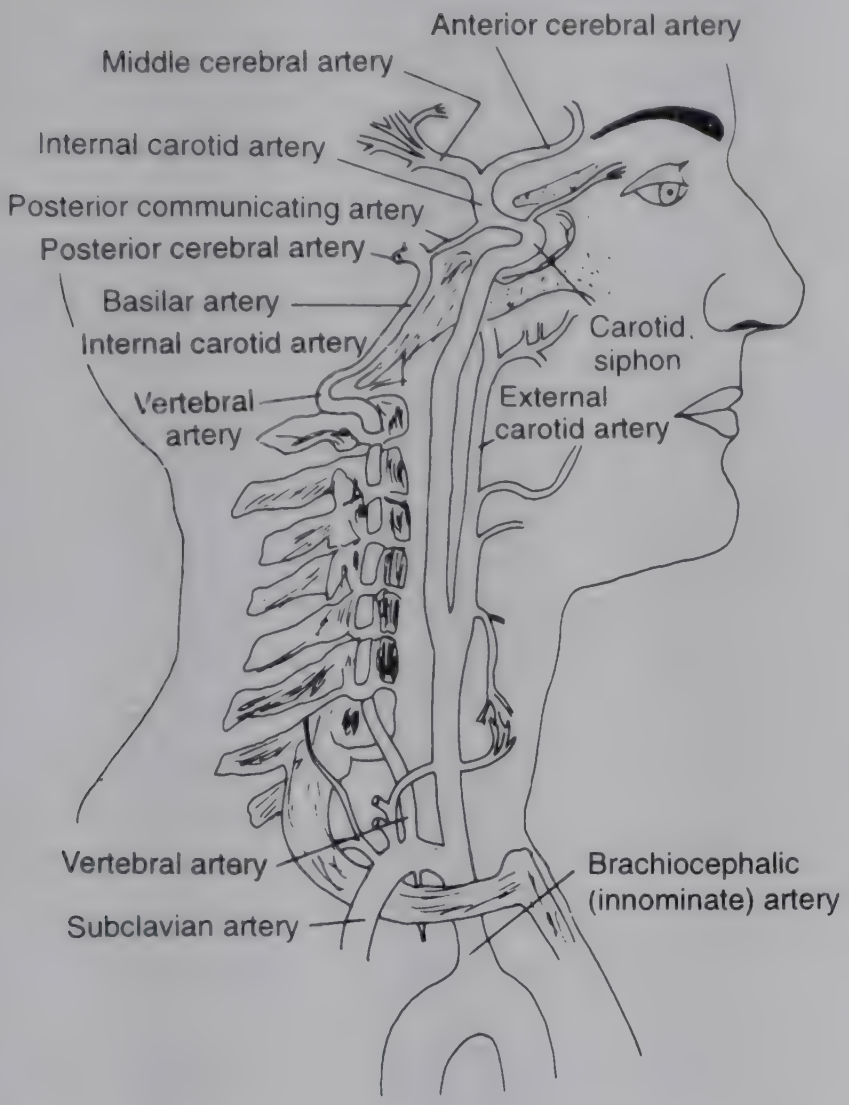

Fig. 10 : The side of the neck (carotid and vertibal) and ascending arteries to the brain.

large arteries to supply the brain (Fig.10). They are the brachiocephalic artery, left common carotid artery, and left subclavian artery. The brachiocephalic artery further divides into the right common carotid and the right subclavian arteries. The right and left common carotid arteries divide into external and internal carotid arteries on either side of the neck (Fig.11). The internal carotid artery on entering the brain divides into two main branches: anterior cerebral artery and middle cerebral artery. Vertebral artery arises from the subclavian artery on each side and enters the cranial cavity through a large opening called the

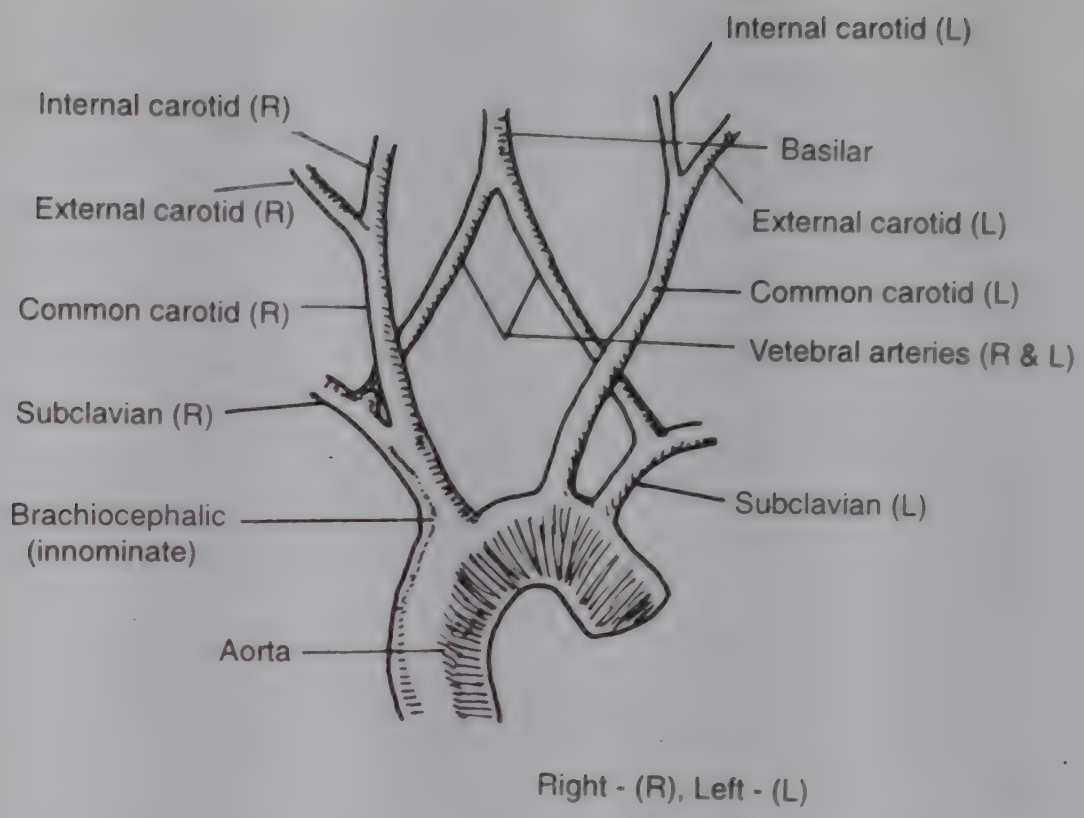

Fig. 11 : The major ascending arteries to the brain arising from aorta, and formation of basilar artery.

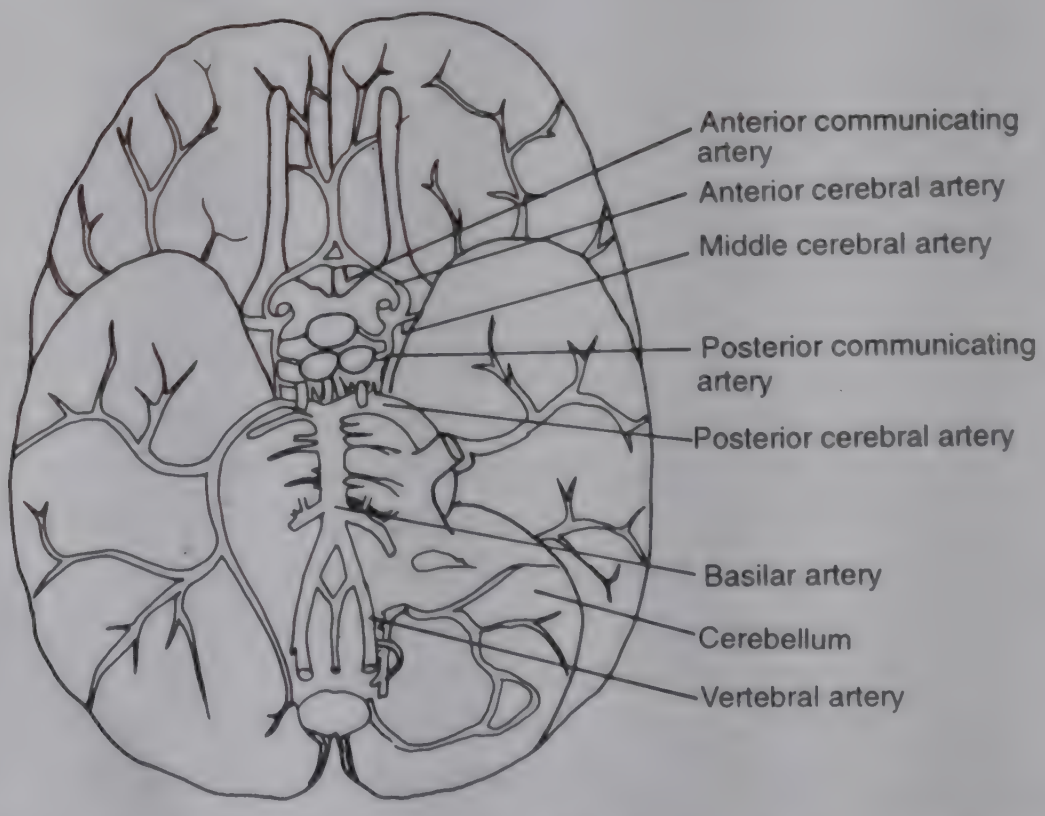

Fig. 12 : The inferior (lower) surface of the brain with arteries at the base of the brain.

foramen magnum (Fig.12). Two vertebral arteries join together at the lower border of pons to form the basilar artery which at the upper border of pons and at the base of the brain bifurcates into two terminal branches, known as posterior cerebral arteries.

At the base of the brain, each anterior cerebral artery, right and left, are joined by a small communicating artery called the anterior communicating artery. Middle cerebral artery on each side joins the posterior cerebral artery through the communicating artery and is named as posterior communicating artery. This hexagonal arterial structure at the base of the brain joining the two carotid system and vertebral basilar system via one anterior communicating and two posterior communicating arteries is named as the circle of Willis (Fig.13). Under normal circumstances the flow of blood remains on the same side, i.e. the right side of the brain is supplied by the right internal carotid artery and its branches and left side of the brain is supplied by the left internal carotid artery and its branches. The vertebral-basilar system supplies to the brain stem, cerebellum,

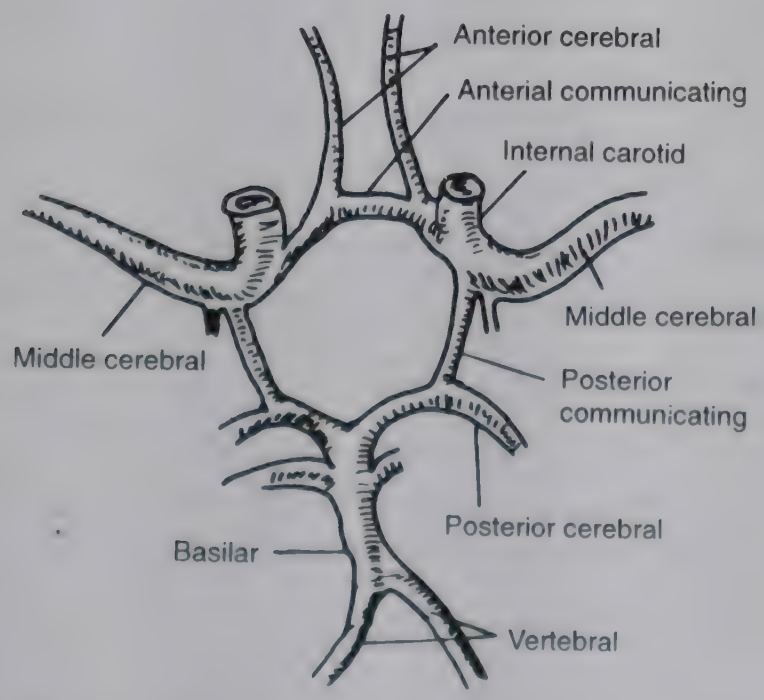

Fig. 13 : The formation of the circle of Willis.

occipital lobes and interior surface of the temporal lobes. About 300 to 400 ml of blood flows each minute into each carotid system and about 200 ml into the vertebral-basilar system. Most of the blood of the internal carotid artery passes through the middle cerebral artery.

As the arteries go deeper into the tissue, the size of these vessels becomes smaller and smaller. Small arteries are called arterioles. The pattern of arterial supply basically is the same for the entire brain. Small arteries and arterioles give off innumerable unnamed perforating branches that penetrate the substance of the brain. Most of the perforating vessels have very few interconnections. Blood flows from arterioles to capillaries for exchange of oxygen and other metabolic products. Deoxygenated blood with metabolic end products flows out in the veins (Fig.14). Venous blood through a network of veins and sinuses returns to the larger veins in the neck. From their it flows into the right side of the heart through superior vena cava. Blood from right side of the heart goes to the lungs for oxygenation.

Most of the large and medium-sized arteries give rise to branches and are named according to the area of the

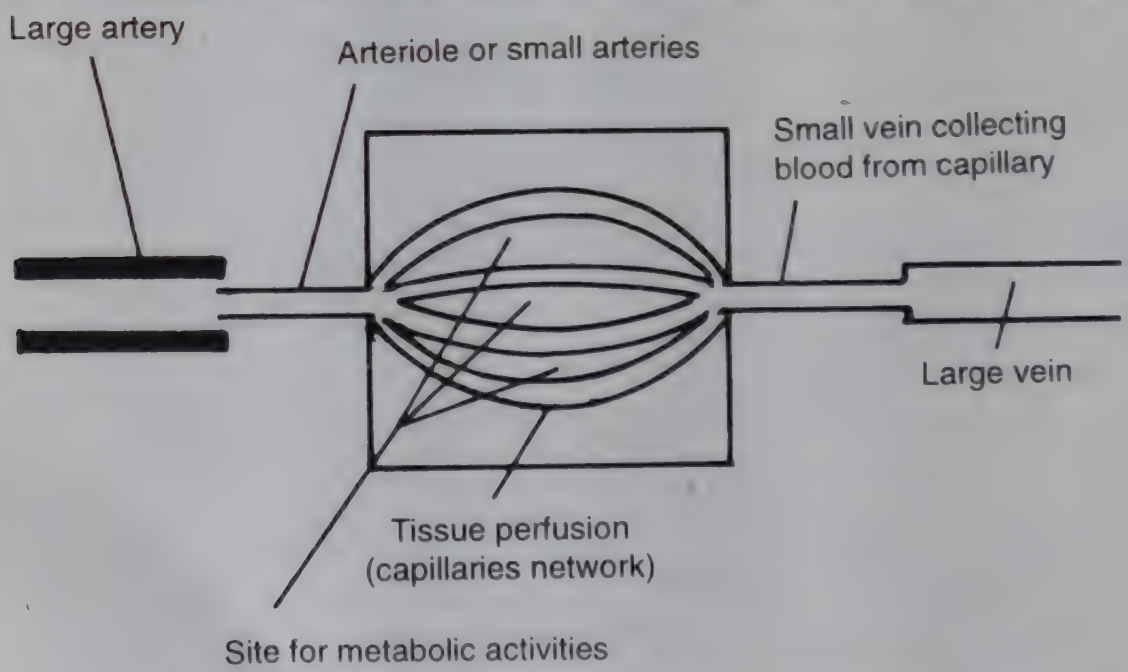

Fig. 14 : The course of normal blood flow from arteries to capillaries and to veins to subserve its function, i.e. to deliver oxygen and other metabolites to tissues.

brain supplied by them. In order to understand the various clinical syndromes (Fig.3, 4, 12) it is important to have some knowledge of the functional areas of the brain and its blood vessels. It is equally important to appreciate that there exists a fair amount of variability in the number of branches, division of arteries and areas of the brain supplied by them. This is one of the explanations of the variability seen in clinical syndromes after a stroke. There is a vast network of blood vessels in the brain. A significant portion of this network remains dormant or inactive and does not participate in the circulation (Fig.15). But whenever there is a

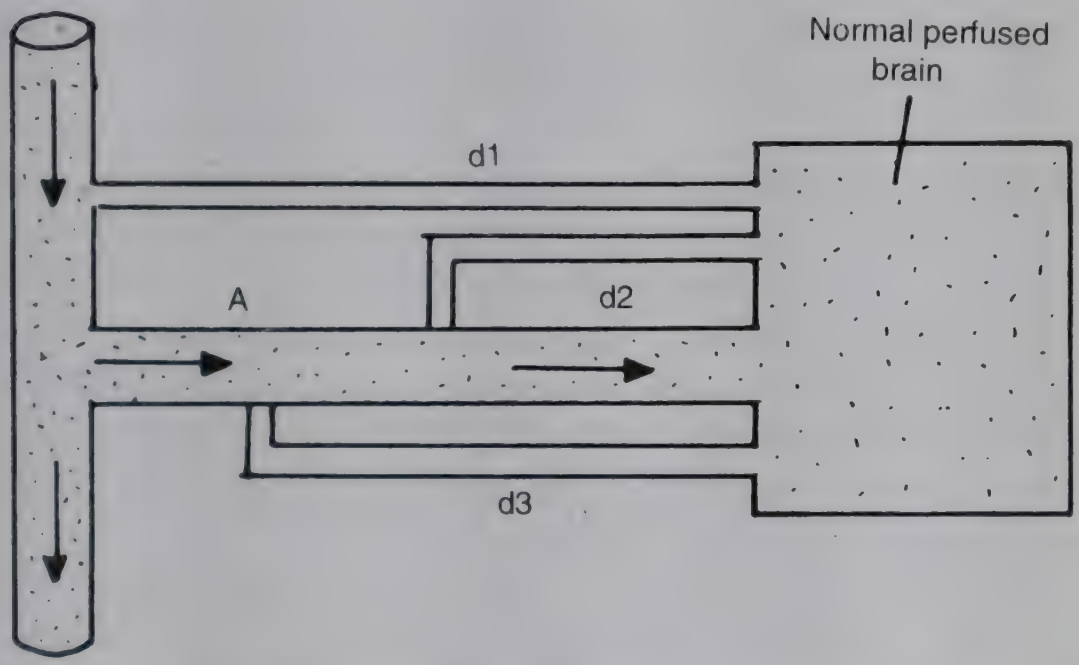

Fig. 15 : The diagramatic presentation of arterial perfusion of the brain parenchyma by the artery A.

demand, due to obstruction in the outflow vessels the dormant network opens up and participates in the cerebral circulation and perfusion (Fig.16). Similarly there exist interconnections and collateral circulation between the branches of internal carotid artery and external carotid artery, between the branches of vertebral and external carotid artery, and between the branches of external carotid and subclavian artery. The circle of Willis at the base of the

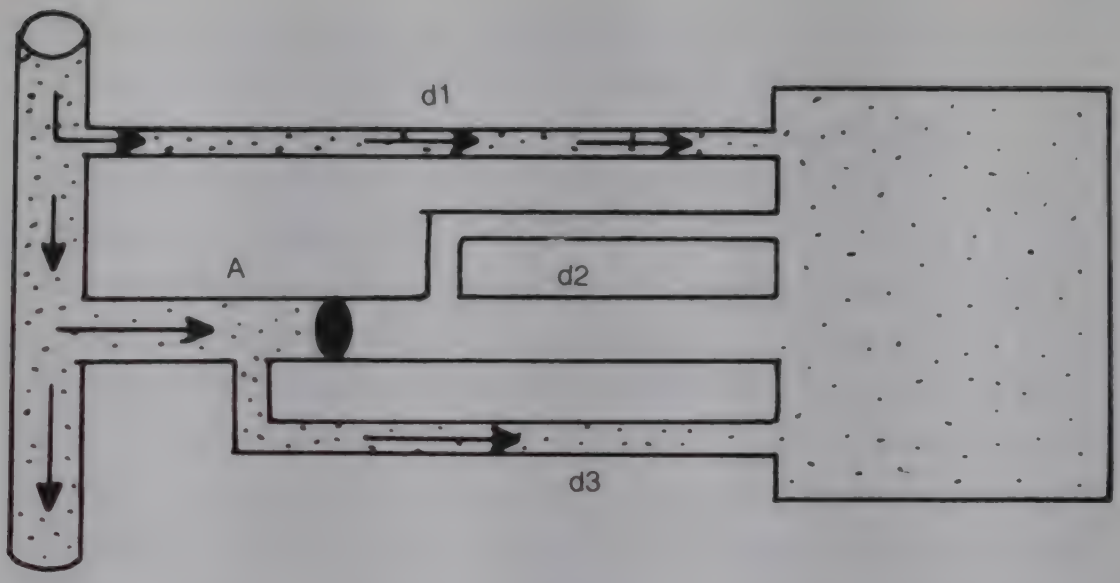

Fig. 16 : The process of normal perfusion by opening up of the dormant vessels once the main perfusion artery is blocked.

brain is an important interconnection point between internal carotids and vertebral-basilar system. There are also effective connections between anterior, middle and posterior cerebral arteries. Persistence of embryonic channels into adult life also serves the same purpose. The function of the collateral channels is to restore the blood supply (Fig.16) and to reduce or limit the damage to the nervous system and hasten the functional recovery.

3

PHYSIOLOGY AND PATHOPHYSIOLOGY

The adult human brain weighs about 1.2 kg (1,200 grammes). The average human body weight is about 70 kg. The ratio of adult human brain weight to body weight is 1:60. Every part of the brain must have continuous blood supply for its function. Interruption of blood supply to any part of the brain results in loss of a specific function. Regardless of the needs of other organs and with only one-sixtieth of the body weight in the adult, the brain gets 16 per cent of the cardiac output (oxygenated blood pumped by the heart). Even in adverse conditions the brain tries to get this blood supply through a regulatory mechanism at the cost of other organs. In infancy and childhood the brain needs even more blood supply and gets about one-third of the cardiac output. Thus, as far as the blood supply is concerned, the brain is the most demanding and vulnerable organ in the body. This demand is much more in infancy and childhood, and if not met with may lead to severe brain damage.

The volume of the human adult brain is about 1,500 ml. It contains at any time about 100 to 150 ml blood and 100 to 150 ml cerebrospinal fluid. The adult brain requires 500 to 600 ml of oxygen and 75 to 100 mg of glucose each minute. To meet these requirements the adult brain is supplied with 55 ml of blood per 100 gm of brain per minute. Roughly 800 to 1,000 ml of oxygenated glucose-laden blood circulates through the brain each minute. The critical blood flow value is 20 ml per 100 gm of brain per minute. There is irreparable brain damage if the supply is

below 15 ml per 100 gm of brain per minute. If the supply is between 15 ml and 20 ml, brain damage can be prevented by restoration of the blood supply quickly. If the blood supply to the brain is interrupted for only 30 seconds, metabolism of the nerve cell changes: in two minutes it ceases; and after five minutes the cell begins to die. Of the brain cells, neurons are the most vulnerable to lack of blood supply because of their negligible energy stores. Neurons require continuous and abundant supply of oxygen and glucose. Of the total glucose entering the neurons, 85 per cent is metabolised into carbon dioxide (CO_2) and water, and 15 per cent to lactic and pyruvic acids. All the metabolic products are removed in the venous outflow.

Autoregulation

All body functions are represented in the brain. Different functions are called into play at different times. Those functional areas that are working at a particular time require more blood supply than the inactive areas. Demand dictates delivery of blood supply. Thus, for example, an orator needs more blood supply to the speech area during oratory; a writer while writing will require more blood flow to the area involved in writing. Nature has provided an autoregulatary mechanism so that the blood supply to the brain is kept to the demand despite a wide range of blood flow. This autoregulation occurs at the level of the circle of Willis, which increases blood supply to one of the either hemispheres or by regional blood flow to any specific area of the brain. Blood vessels of the brain dilate if there is fall in blood pressure, and constrict if there is rise in blood pressure. The lower limit of cerebral autoregulation and the lowest tolerable blood pressure in humans with normal blood pressure are stated to be 50 to 70 mm of mercury and 35 to 40 mm of mercury, respectively. Studies have shown that chronic hypertension (high blood pressure) produces changes in the blood vessels supplying the brain which

produce a shift in cerebral autoregulation to higher levels. In severe hypertension these limits can be as high as 85 to 150 mm of mercury and 55 to 85 mm of mercury, respectively. These higher levels of autoregulation in patients with high blood pressure can be reverted to normal range by appropriate therapy to reduce blood pressure. Thus reverting to normal range in patients with high blood pressure leads to reduction in cerebral blood flow. Therefore caution should be exercised while reducing the blood pressure in strokes caused by high blood pressure.

Autoregulation ensures adequate cerebral circulation in a wide range of blood pressure variation. But acute elevation of blood pressure causes dilatation of arterioles, increased cerebral blood flow and increased permeability. These result as a consequence of damage to the inner lining of blood vessels and smooth muscle which in turn lead to reduced responsiveness to stimuli that constrict the blood vessels, and reduced ability for autoregulation. These changes also lead to a decreased wall thickness and greater dysfunction of the blood-brain barrier with increased permeability. As a result, small balloon-like structures called Charcot Bouchard aneurysms are formed at the weak points along the damaged wall of the blood vessels, which bleed when ruptured.

Blood Flow Velocity

Each internal carotid artery supplies approximately 300 to 400 ml of blood per minute. About 200 ml of blood flows through the vertebral-basilar artery each minute. The blood flows faster in the carotid circulation than in the vertebral-basilar system. In the capillary bed the blood stays only for about 2 seconds in each cycle and within this brief time metabolic exchange must take place.

There is no difference between pressures among the carotids and the vertebral circulations because normally the circle of Willis equilibrates them unless anomaly or disease

cause them to fluctuate independently. Narrowing of an artery alters pressure at the far end, flow velocity, and volume of flow. There are changes in blood flow that occur in the awake and the sleeping stages. There is marked increase in blood flow during rapid eye movement (REM) sleep. On the other hand, cerebral blood flow and metabolism are depressed during coma. Thus there is a close coupling between blood flow and metabolism. There is increased cerebral blood flow whenever there is increased metabolic requirement. Contrary to common belief, the blood supply to the brain is neither increased by gravity by standing on one's head nor is it reduced by the ingestion of a heavy meal.

Brain Metabolism

As the brain does not store glucose in any significant quantity and cannot shift to an effective anaerobic cycle for an alternative source of energy, effective blood circulation has to be maintained for proper brain function. If the blood flow is cut off even for 10 seconds, lactic acid produced accumulates and neuronal activity is impaired. Neurons and glial cells remain alive but functionless, for as long as 30 minutes, before cellular death occurs. If blood flow is impaired but not completely cut off, neurons can be functionless but viable for 6 to 8 hours. With the reduction of the cerebral blood flow, the brain extracts more and more oxygen to meet the requirement until a critical level is reached. Further reduction below this level does not allow extraction of enough oxygen as necessary and leads to brain damage. With normal brain circulation 15 per cent of the glucose is metabolised to lactic and pyruvic acids generating heat in the process. With reduced blood flow to the brain and in the presence of high cerebral glucose level there is increased glycolysis and increased accumulation of lactic acid. High lactate levels (16 mmol/kg or higher) leads to cerebral infarction (death of brain cells). Thus good

control of the high blood glucose is necessary to reduce the risk of cerebral infarction in stroke patients.

One of the end products of the brain metabolism is carbon dioxide (CO_2). This finds its way to the venous capillaries and is eventually removed from the blood by the lung. Before CO_2 reaches venous capillaries it causes dilatation of the arterioles and increases the blood flow. Arterioles also dilate in response to other metabolic products and hydrogen ions. The pressure of carbon dioxide in the arterial blood is maintained normally at about 40 mm of mercury. If it falls then the arteries constrict and blood flow is reduced. Pressure of oxygen in the arterial blood is normally about 80 mm of mercury. Effect of oxygen is almost the opposite of those described for CO_2. A sharp decrease in the oxygen pressure leads to some dilation of the arteries, but this is seldom enough to maintain constant oxygenation. On the other hand, because of autoregulation cerebral arterioles do not respond directly to pharmacological agents. Therefore such agents do not have a clinically significant effect on cerebral blood flow.

Pathophysiology

Reduction in the brain blood flow sufficient to interfere with brain function leads to ischaemia. Ischaemia leads to brain hypoxia but these two terms are not interchangeable. Hypoxia means low oxygen content in the blood supply to the brain. Ischaemia implies not only reduced delivery of oxygen and glucose but also failure to remove metabolic end products and thereby accumulation of these products at the local site. The effects of hypoxia are aggravated in the presence of hypotension (low blood pressure) or ischaemia. Cerebral oxygen consumption is reduced with the reduction of glucose in the blood. Ischaemia leads to brain infarction with varying loss of autoregulation and carbon dioxide responsiveness. The local area of ischaemia at times is surrounded by an area of excess blood. This results either

from canalisation or a clot moving away from the site of formation. This relative excess of blood supply that exceeds metabolic needs is called 'luxury perfusion'. Arterioles in the ischaemic area dilate maximally under the influence of carbon dioxide, hydrogen ions and lack of oxygen. If the vasodilator (a drug that dilates blood vessels) is used, arterioles in the ischaemic area do not dilate further, but arterioles in the surrounding normal area dilate and blood flows from the ischaemic dilated arterioles into the normal brain arterioles. This deprives the ischaemic area further and this phenomenon is called 'steal'. Therefore, administration of vasodilators for acute infarction is ineffective and may be, harmful. Hyperventilation leading to decrease in the pressure of carbon dioxide in blood, on the other hand, causes constriction of normal arterioles but has no effect on the maximally dilated arterioles in the ischaemic area. Thus blood moves from the normal to the ischaemic area.

4

MECHANISM

The term of cerebrovascular disease encompasses any disease of the brain resulting from a pathologic process of blood vessel. There is a widespread elaborate network of vessels in the brain. The brain receives the blood through the large arteries in the neck (Fig.11) which are relatively superficial and are at risk of external compression. But as the arteries reach the brain proper they become smaller and smaller in diameter and are called arterioles. Within the brain, blood passes from arterioles to capillaries for oxygen and metabolic exchange (Fig.14). Deoxygenated blood with metabolic end products is carried off by veins from capillaries to larger veins to the right side of the heart and then to the lungs for oxygenation. Pathological processes involving the blood vessels (Fig.17) may be (i) lesions outside the vessels, (ii) lesions of the vessel wall, and (iii) lesions of blood flowing in the vessels.

Lesions may involve either the arteries, capillaries or veins. The vascular system can be involved at any level, namely heart, neck or brain. Different diseases have their own characteristics and one should know about these disease processes for better understanding.

Compression of the vessels from outside can occur in the neck or within the skull. Any space-occupying lesion such as a tumour, haematoma, infective granuloma and herniated brain, etc., can compress the blood vessel. Similarly any injury in the neck or hanging can compress the internal carotid or vertebral arteries. Instead of vessels getting compressed, some vessel abnormalities like dilated

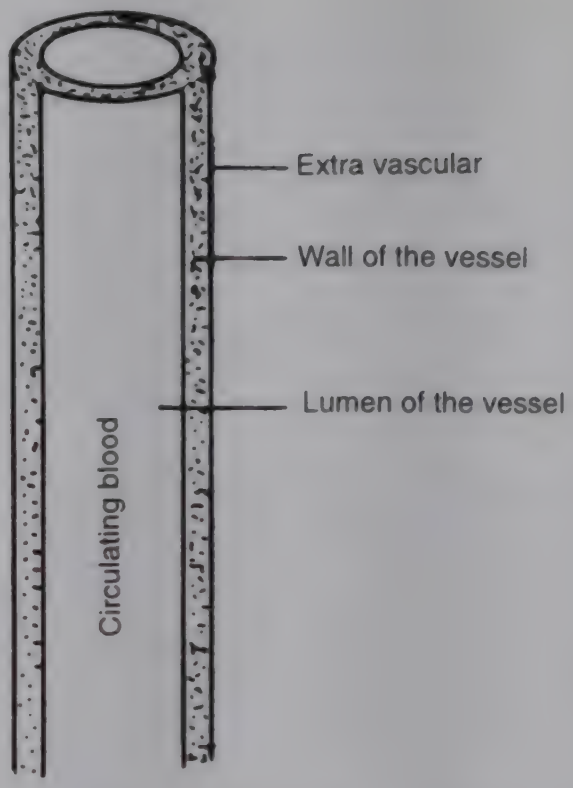

Fig. 17 : Various sites of the blood vessel where the disease process can start.

and hardened arteries, giant aneurysms, and large arteriovenous malformations may cause local pressure effect on the nearby brain tissue and blood vessels, and result in brain dysfunction.

Arteriosclerosis (thickening) and atherosclerosis (hardening) may involve the wall of the arteries in the neck or brain. Atherosclerosis is more common in larger arteries while arteriosclerosis is mostly seen in the perforating or end-arteries and arterioles deep in the brain. Formation of Charcot Bouchard aneurysm in hypertension leads to haemorrhage in thickened arterioles. Atherosclerosis is caused by lipid deposition and plaque formation (Fig.18). Inflammatory diseases may also involve the arterial wall, a condition known as arteritis. Infection of veins and sinuses resulting in clot formation may lead to strokes.

Other predisposing factors include disorders of the cavity of the blood vessel or the blood constituents that flow continuously through the cavity. Increased viscosity of the

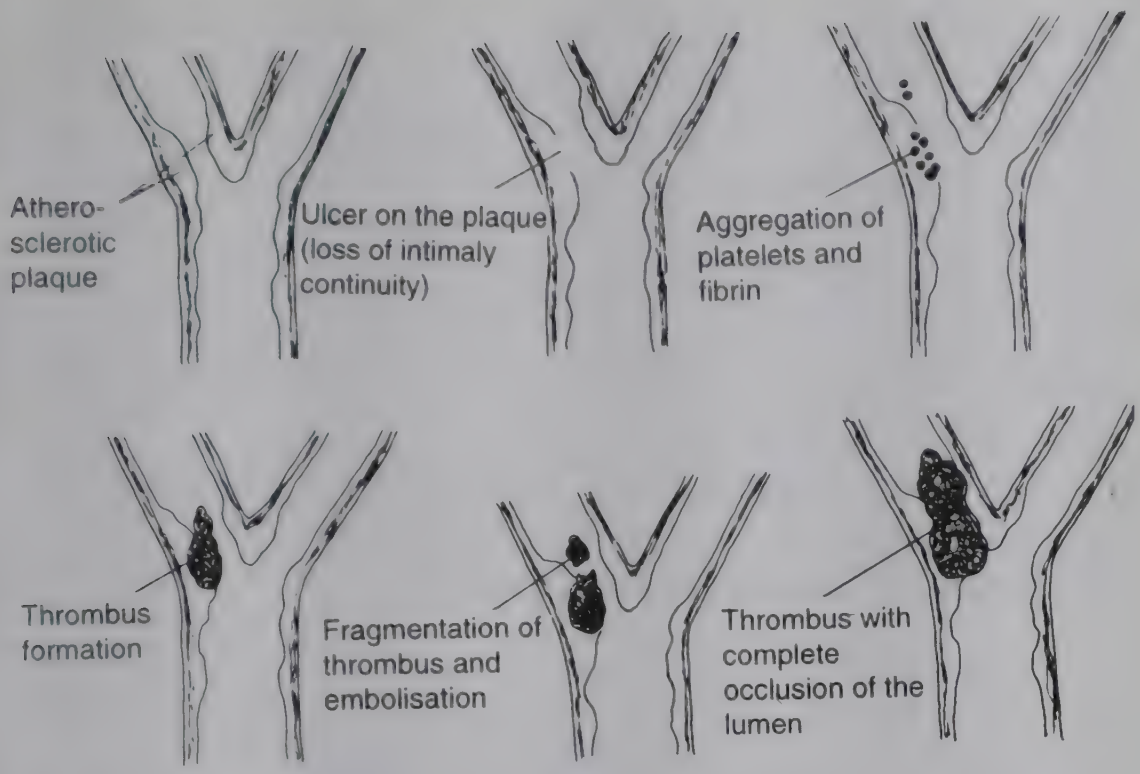

Fig. 18 : Various stages of atherosclerosis and occlusion by the formation of thrombus in the vessels in the neck. Thrombus may get detached and leads to embolus.

blood slows down the circulation and may block the cavity. Increased adhesiveness of the blood platelets leads to the formation of clots. Diminished oxygen carrying capacity of the blood as in anaemia may also result in cerebral dysfunction. There are some other conditions, with undetermined causes, that are associated with cerebro-vascular diseases.

The pathological changes that occur in cerebro-vascular diseases can be broadly divided into thrombosis, embolism and rupture of the vessel. In many clinical situations it is difficult to differentiate between thrombosis and embolism. Therefore the term thromboembolism is more often used in clinical practice. Formation of blood clot in the cavity of the vessel gets bigger and bigger in size till it occludes the lumen. Before complete occlusion there is narrowing of the arterial lumen.

Changes in the brain tissue that occur following changes in blood vessels are of two types : (i) ischaemia (deficiency

of blood) with or without infarction, and (ii) haemorrhage. Changes in brain tissue can be seen at autopsy in patients who did not clinically have either ischaemia or haemorrhage. Infarction is the descriptive term in pathology in which destruction or disruption of the brain tissue occurs as a result of impaired blood supply (Plate I). But impaired blood supply may not always cause infarction. The development of infarction depends upon the duration of impairment of blood circulation.

In haemorrhage, blood leaks from the vessel, usually arterioles and end arteries, under high pressure. The blood leaks directly into the brain (Plates II, III, IV, V; Fig.19). The

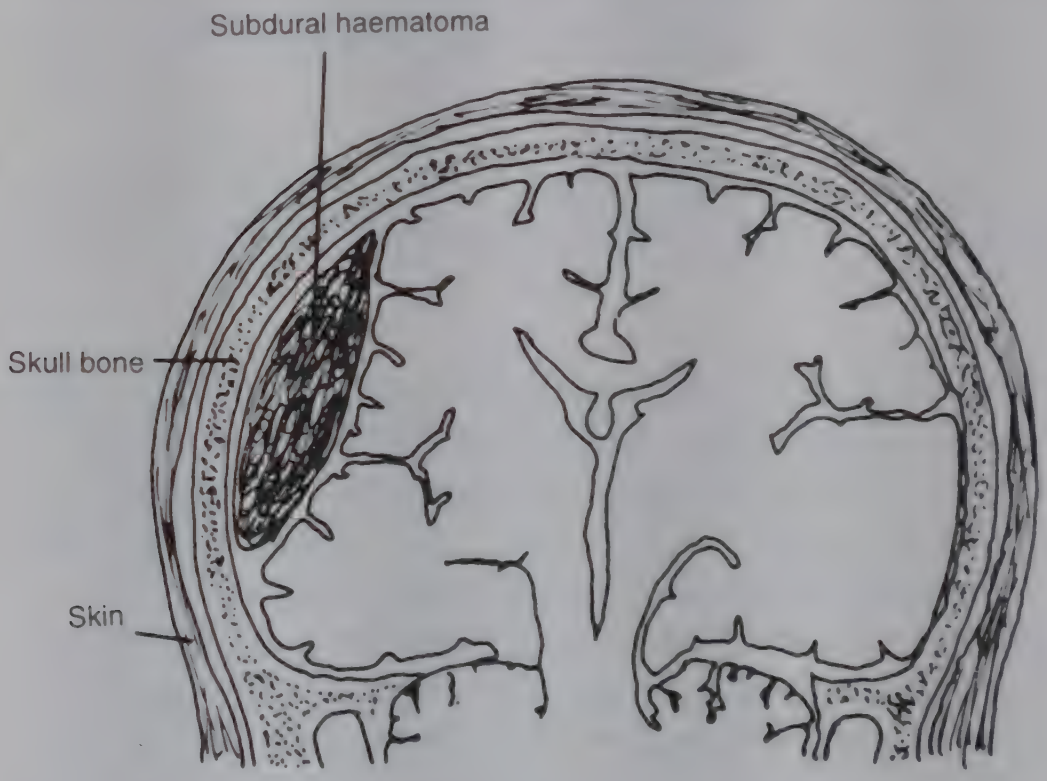

Fig. 19 : The subdural haematoma and its anatomical relationship to skin, bone and brain. Brain can be significantly compressed.

leaked blood does not remain confined to any vascular territory and may extend to any space or direction of the least resistance. The mass of clotted blood causes disruption of the tissue (Plates II, IV) and puts pressure on the surrounding brain leading to neurological dysfunction. The

primary haemorrhage can lead to secondary haemorrhages (Plate VI). The mortality and morbidity from haemorrhage depends primarily upon the size and site of blood clot. If the patient survives then the blood clot slowly disintegrates and is absorbed over a period of weeks and months. Haemorrhages by and large are not repetitive.

5

RISK FACTORS

Recognition of risk factors is one of the most outstanding contributions of research on cerebrovascular diseases in the last few decades. These factors are known to increase the subject's susceptibility to stroke. There is good amount of evidence to indicate that there is a decline in the incidence of stroke following management of treatable risk factors. Since an effective and curative treatment for stroke is not so far available, efforts should continue to concentrate on prevention. There are treatable and untreatable risk factors.

Untreatable Risk Factors

- *Age*: Aging remains the biggest and most serious risk factor for stroke. Incidence of stroke increases with age. It is believed that morbidity and mortality from stroke doubles every decade after the age of 50 years.
- *Sex*: Women are protected from stroke before menopause. Men aged under 60 to 65 years have a 30 per cent higher incidence of stroke than women.
- *Race*: Black communities have been found to have a higher mortality rate from stroke in comparison to whites.
- *Ethnicity*: Japanese and Chinese in the north-eastern region of mainland China show a higher incidence of stroke. Japanese immigrants to Hawaii and California have been found to have a lower stroke mortality

than their brethren in Japan. This suggests that racial characteristics if any are amenable to modification by environment.

- *Season*: Stroke incidence is higher in low ambient temperatures. Peak months for stroke are winter months.
- *Genetic*: There is no doubt about the genetic predisposition to strokes in some families. There is definite history of higher cases of strokes in some families both in vertical and horizontal pedigrees.

Treatable Risk Factors

- *Hypertension*: The most important treatable risk factor for stroke is, of course, hypertension. It appears that systolic blood pressure is as important as diastolic pressure in the causation of cerebro-vascular disease. The higher the blood pressure, the greater is the risk. A sudden increase of diastolic blood pressure more often leads to cerebral haemorrhage. Even an isolated increase of systolic blood pressure is a risk factor and therefore should not be ignored. The decline in stroke incidences in recent years has been mainly due to long term control of hypertension. This has reduced the incidence of both brain infarction and haemorrhage.
- *Heart disease*: Heart disease (particularly congestive heart failure), coronary atherosclerosis, and arrhythmias (particularly atrial fibrillation) greatly increase the probability of a stroke. Heart disease accounts for the largest number of deaths among stroke survivors.
- *Diabetes mellitus*: Diabetes is also a significant risk factor especially amongst women. The risk of infarction is doubled at blood sugar levels above 160 md/dl. Diabetes hastens the hardening process in both large and small arteries.
- *Hyperlipidemia*: Increased lipids (fats) in blood also constitute a risk factor for stroke. There are some

controversies here. The role of total cholesterol and lipoprotein fractions remains controversial.

- *Obesity*: Obesity is not an independent risk factor for stroke but it aggravates the hardening of arteries and high blood pressure and ischaemic heart disease.
- *Smoking*: Smoking is also regarded as an independent risk factor for stroke. Incidence of stroke has been found to be higher in those who smoke regularly and heavily.
- *High haematocrit*: Increased haemoglobin level is another risk factor for stroke. Incidence of stroke is higher in patients with abnormally high haemoglobin level than in anaemic patients.
- *Transient ischaemic attacks (TIA):* Patients with transient ischaemic attacks are at tenfold increased risk for stroke compared to normal individuals. About 35 per cent of those who had suffered a TIA could expect a stroke within the following five years. Nearly 50 per cent of these could have their stroke within one month of their first TIA.
- *Stroke*: A prior stroke is now regarded as a major risk factor. The risk of a recurrent stroke is many times more than that of a first stroke.
- *Oral contraceptives*: An additional risk factor for women is oral contraceptives.
- *Alcohol*: Intake of alcohol is a risk factor for men. The degree of risk depends on the amount of alcohol consumed.

A number of studies have confirmed that by controlling hypertension the risk of stroke can be reduced significantly. Therefore it makes sense to make every effort to avoid, control or cut down the risk factors. However, it should not be taken for granted that we can eliminate all strokes by controlling risk factors. Many people not exposed to any kind of treatable risk factors still suffer stroke. It is believed at present that control of risk factors can

prevent only a limited percentage of strokes because there are many untreatable risk factors too.

Preventive measures should start as early as possible but ideally one should start at school. There are some modifiable risk factors which should be impressed upon the young adults at school age. Facts like smoking, alcohol, diet, exercise and oral contraceptives should be brought to their attention. If we succeed in conveying to the young people the risks involved for the development of stroke later in life, we shall have achieved significant success in the prevention of stroke.

6

ATHEROSCLEROSIS AND ARTERIOSCLEROSIS

Arteries in young persons are soft and have a fair degree of elasticity. But they tend to thicken progressively and become relatively stiff and lose elasticity with aging. This is because of a gradual loss of elastic tissue and its replacement by fibrous tissue in the arterial wall. This pathological change is called arteriosclerosis. It is essentially a non-inflammatory degenerative disease that affects all arteries in the body. Another pathological change seen in arteries is a thickening of its walls. The major cause of arterial thickening is the deposition of fat or lipids within the arterial wall. This is called atherosclerosis. The fat deposition also reduces the elasticity of the arteries. Atherosclerosis is a gradually progressive disorder. It is known to begin early in life. Its origin can be traced right from the first decade of life. However, the progression of atherosclerosis is generally slow and it does not become symptomatic until the middle age.

Atherosclerosis afflicts almost everybody. The major arteries of the body like aorta, arteries in the neck, cerebral arteries, coronary arteries, renal arteries and arteries of the lower limbs are the ones most severely and frequently affected by atherosclerosis. The severity of atherosclerosis and involvement of different arteries varies from person to person.

Atherosclerosis may begin from an injury to the arterial wall. Lipids, mostly cholesterol, are then deposited in the smooth muscle cells of the middle layer of the vessel

wall. Fatty streaks are visible early in the course, i.e. in the first or second decade. Around third decade plaques are visible. Plaques are formed by the increased number of smooth muscle cells and fibrous tissue around the cholesterol deposit in the internal lining of the artery. These plaques enlarge in size and number and coalesce, and there can be a deposition of calcium. Later, clot may be formed over the roughened elevated surface of the plaques by the aggravation of slow moving adhesive platelets (Fig.18). The clot may gradually increase in size, leading to a narrowing of the artery gradually or may block it altogether. The clot may also get detached from the site and may move to a distant part in the circulation until it causes a blockage or gets fragmented or dissolved. Thromboembolism is a major complication of atherosclerosis that leads to ischaemia and infarction. Arterial wall overlying the plaques gets weakened, resulting in abnormal dilation (Charcot Bouchard aneurysm). Haemorrhage may occur from these weak aneurysmal dilatations.

There is a wide range of variability in the extent, severity and distribution of atherosclosotic lesions in different arteries and in different people. Persons with milder forms of atherosclerosis may never have any symptoms for a long time until the advanced age when severity of the lesions becomes sufficient to cause symptoms. Those who have severe or rapidly progressive atherosclerosis may have symptoms in their thirties or forties.

Total fat or lipid contents of the blood have several components. Cholesterol is one of the important lipoproteins which circulates in the blood. There are three major classes of lipoproteins in the blood: very low density lipoproteins (VLDL), low density lipoproteins (LDL), and high density lipoproteins (HDL). The LDL makes up 60 to 70 per cent of the total serum cholesterol while HDL usually constitutes 20 to 30 per cent. The raised serum cholesterol, predominantly the LDL cholesterol, is a strong, independent and consistent causal factor for atherosclerosis,

ischaemic heart disease and stroke. The protective role of HDL cholesterol is now well documented. A high HDL/LDL ratio means less risk of coronary artery disease. Therefore LDL-cholesterol should be kept below 130 mg/100 ml while HDL-cholesterol should not be less than 30 mg/100 ml and preferably be higher than 50 mg/100 ml. The cholesterol level in blood depends partly on the fat and cholesterol content of the diet. Other factors such as obesity and physical inactivity also play a role. After a stroke, diet with low LDL-cholesterol and high HDL-cholesterol contents should be the first line of management. Linoleic acid and oleic acid are effective in reducing the LDL-cholesterol level when either is substituted for saturated fatty acids in the diet. Several drugs now available, which can reduce serum cholesterol, should be supplemented if necessary.

7

TRANSIENT ISCHAEMIC ATTACKS

Transient ischaemic attacks (TIA) are episodes of temporary nature which manifest as sudden impairment of some function, focal neurologic deficit of vascular origin, which are restored completely in less than 24 hours, and leave no permanent damage. TIAs are divided into two categories on the basis of the circulatory system involved. The common symptoms in carotid TIAs are weakness of muscles, numbness, difficulty in speech or other language dysfunction, blindness in one eye, etc. The common symptoms in vertebral-basilar TIAs are vertigo, quadriparesis, difficulty in speech, lack of co-ordination, double vision, difficulty in swallowing, numbness, loss of consciousness, visual field defects, etc.

The frequency of TIAs is very variable. The data are based on information collected from patients and their relatives and are likely to be inaccurate. Some patients tend to dismiss these because of their transient nature. There is no laboratory test or simple investigation to confirm clinical suspicions. According to some, only 10 per cent of all strokes are preceded by a transient ischaemic attack. Most of the strokes occur without any warning and with catastrophic suddenness. Men are more affected then women, and the onset occurs most frequently in the 50 to 70 year age group. This is the age group in which other risk factors for strokes are also present. A majority of TIAs occur in the carotid system and about 10 per cent in the vertebral-basilar distribution, and very rarely in both systems. TIAs often recur but the number of attacks in an individual

patient is variable. One-third of patients suffer only a single attack.

Transient ischaemic attacks were believed to leave no permanent damage when their significance was recognised in clinical practice in early 1960. This is now no longer correct. There is now mounting evidence, provided by CT, MRI or SPECT, that almost every TIA results in some neurological damage even if, in clinical terms, there are no signs of any disturbance after 24 hours. Traditionally a distinction has been made between TIA, reversible ischaemic neurological deficit (RIND), and minor stroke. RINDs are defined as episodes in which the neurological deficit last longer than 24 hours but is fully resolved within a week or two. Minor strokes are defined as episodes in which only minor or small neurological deficit persists permanently in spite of a good clinical recovery. It is reasonable, therefore, to regard TIA, RIND and minor strokes as a continuous spectrum of clinical conditions, any of which can lead to a major stroke or infarction.

Pathogenesis

There are many mechanisms of TIAs. Atherosclerosis with thromboembolism is the most important and common cause of TIA.

The brain meets its blood supply and metabolic demand by the mechanism of autoregulation—a unique feature for brain. This is true for the brain as a whole or different parts of the brain at different time of activity. If the blood circulation is not sufficient in amount and in time, brain function may be impaired leading to TIA. There are more than one way in which blood circulation is impaired. Systemic blood pressure is very important in maintaining cerebral circulation. In practice, however, low systemic pressure is relatively a rare cause of TIA. Systemic hypotension may result from irregular heart beat or blood loss. Haematological causes increasing the blood viscosity

or leading to poor oxygen carrying capacity can also lead to TIA.

TIAs may also be caused by rapid neck movement due to external compression of an artery. Turning the head to one side leads to compression of vertebral artery on the opposite side and stretching of the artery on the same side. This is more likely in patients who have cervical spondylosis or other cranio-vertebral anomalies in the neck.

Formation of blood clots is an important and major cause of TIAs, especially in the carotid circulation. These tiny clots cause blockage of the arteries and lead to temporary disruption in blood supply to the brain. The tiny clots eventually get broken up spontaneously and normal blood supply is restored.

More than one mechanism may be working in individual patients. There may be some other factors like vascular wall abnormality, reversal of blood flow, etc., which may impair the cerebral circulation and lead to TIAs. It is for this reason that the patient needs a complete systemic examination.

The diagnosis of TIA is essentially based on the history of the patient. It is therefore necessary that full clinical examination is done to eliminate other possibilities. Non-invasive tests for assessment of carotid and subclavian arteries in the neck like doppler and real time ultrasound sonography should be carried out in all cases. CT, MRI and MR spectroscopy or SPECT should be done depending upon the available facilities and planning of the therapeutic modalities. Angiography and digital subtraction angiography (DSA) have provided a useful way to study the major vessels in the neck and intracranial vessels.

While planning any therapeutic modalities, two things should be kept in mind. Firstly, there are multiple causes of TIA, although thromboembolism is the commonest and most frequent cause. Secondly, TIA is the precursor of ischaemic stroke. It provides a warning to the patient to take necessary preventive steps. Within 5 years of a TIA, 35

per cent of TIA patients are likely to have a full-scale stroke. Even more significant, 50 per cent of these may suffer a stroke within one month of the their first TIA. One-third who do not suffer a stroke, will continue to have recurrent TIAs,in the remaining one-third TIAs cease spontaneously. There is no way one can predict in which group a particular patient falls. TIAs in the vertebral-basilar artery are more benign and likely to cease spontaneously in due course of time than the TIAs in the carotid system. There is about 20 to 25 per cent mortality in TIA patients during a 5-year follow-up period. However, the majority of these fatalities are due to myocardial infarction (heart attack) rather than a stroke. This study aspect has given further insight for evaluation and management of heart conditions in TIA patients. Management of other risk factors, particularly hypertension, is of paramount significance. Ultimate prognosis depends upon the collective care of all these factors.

Blood clots in brain arteries being the commonest cause leading to TIAs, antiplatelet drugs and anticoagulants have been used in treating of TIAs. Aspirin (soluble forms) has stood the test of time and is widely used all over the world. There are some controversies over the dosage but no one disagrees about its effectiveness as a preventive. Some believe that aspirin in a dosage of 100 mg or even less per day is adequate for prevention. Small dose is attractive because of less risk of gastrointestinal side effects. Ticlopidine, a new drug with minimal advantage over aspirin, has recently come in the market. Ticlopidine can be used in those situations where aspirin is contraindicated or not tolerated. Other antiplatelet drugs like Dipyridamole and Sulphinpyrazone have mostly been given up as these are not found very effective in brain conditions, though still may be effective in heart conditions.

Anticoagulation therapy is very controversial. It requires good monitoring and good cooperation and thus becomes expensive. Anticoagulation therapy carries almost

a 3 per cent annual risk of serious or even fatal haemorrhage. There are also controversies about the kind and dosages of anticoagulant drugs. Mini-dose warfarin therapy is advised by some, but the utility of anticoagulants in prevention or management has not been established even today.

Surgical management of TIA include endarterectomy or removal of the blocked portion of the artery and placing a graft to maintain circulation. This technique has been used in large numbers in some western countries, but there are no clear-cut accepted indications. Endarterectomy is done in two situations, namely (1) to treat acute blockage of the artery by the clot, and (2) to prevent a stroke. The curative surgery should be done within 6 to 8 hours of onset of infarction. There is very high mortality associated with this surgery and a very few hospitals are equipped to undertake such surgery. Prophylactic endarterectomy is done in-between TIAs or after the infarction has stabilised. Prophylactic operation may, however, result in the very events that it is seeking to prevent for a stroke may occur during surgery or following surgery. Balloon angioplasty has attracted attention of late and one has to wait for the results of trials that are currently in progress.

8

CEREBRAL INFARCTION

Cerebral infarction is a term used to describe the brain damage or death of brain cells and vessels. Cerebral infarction results from many different causes. These include ischaemia (decreased blood flow), or a reduction in the rate of removal of metabolic end products. Different causes of infarction such as anoxia (deficiency of oxygen), ischaemia or hypoglycaemia, have their own characteristic features, area of affliction and pathological changes. Ischaemia is the most common cause of cerebral infarction. Ischaemic infarction is caused by insufficient inflow of blood, despite normal glucose and oxygenation that leads to secondary hypoxia, impaired cellular nutrition, accumulation of metabolic end products and heat with resulting cell death.

One of the characteristics of ischaemic infarction is the limitation of pathological changes in the distribution of an artery in the early stages. Secondary changes may subsequently ensue if the disease advances. Thrombus and embolism, collectively called thromboembolism is the commonest and most frequent cause of ischaemic infarction. There may be complete blockage of an artery without any infarction or there may be partial infarction because of partial restoration of blood supply by other nearby vessels (Fig.20, 21). Therefore there are innumerable variations in the extent of infarction and its degree of completeness (Plate VII). The speed of development of the blockage in the artery is important as gradual narrowing of an artery allows time for collateral channels to open. Increased

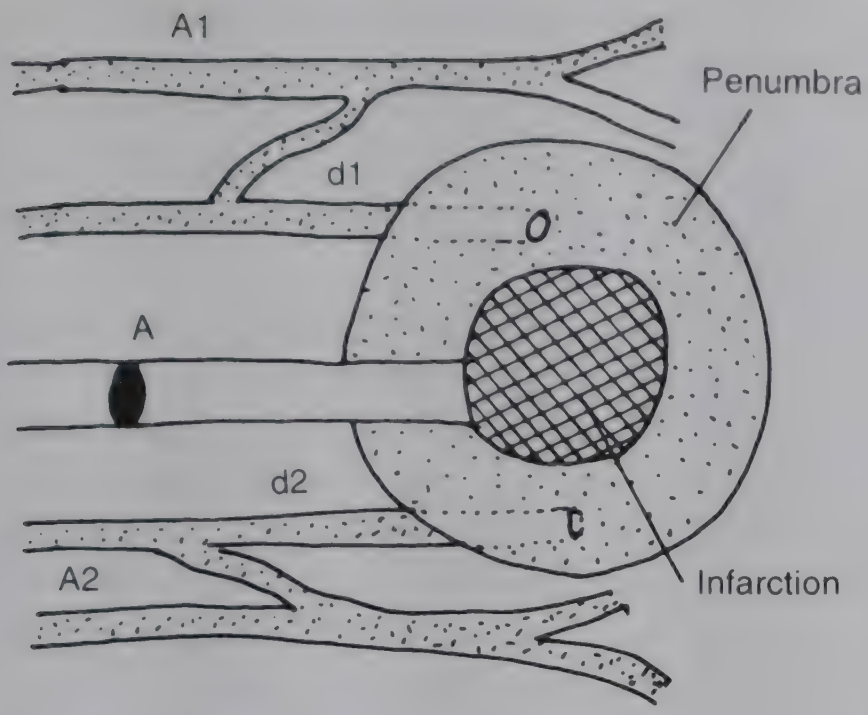

Fig. 20 : The central area supplied by the occluded artery A is infarcted while the periphery is perfused by dormant arteries and is called penumbra. A1,A2 vessels are related and caused hyperaemia at the periphery of penumbra.

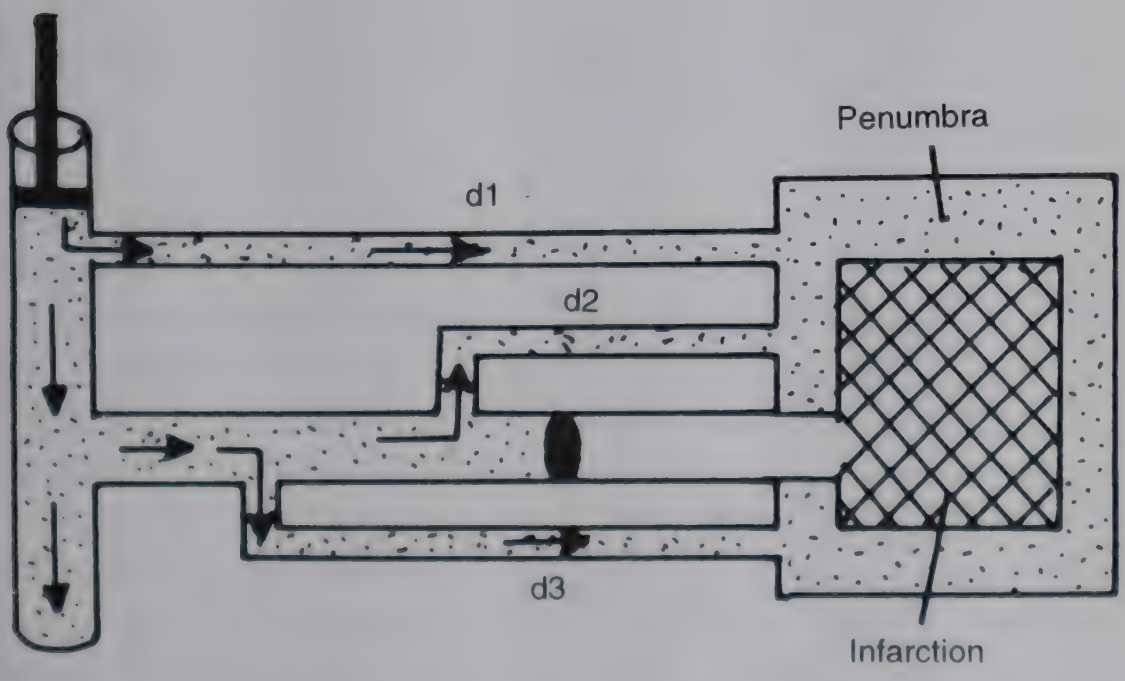

Fig. 21 : Diagramatic representation of the importance of pressure head (i.e. BP) in opening up the collateral and dormant vessels to maintain the perfusion.

viscosity and osmolality of the blood and a high blood sugar level are equally important factors. Reduced oxygen and carbon dioxide levels have a worsening effect. Anomalies of the vessels and previous blockages also have major effect on the extent of infarction and thus clinical outcome.

The dead brain tissue resulting from an ischaemic infarction, swells rapidly, mainly because of excessive intracellular and intercellular water content. This further aggravates the damage of the adjoining brain tissue. There is accumulation of H-ions and lactic acid, other metabolic end products, free radicals, and sodium, potassium and calcium ions in the dead brain tissue which cannot be removed in the outflow venous circulation. There is disruption and disintegration of neurotransmitters (chemicals involved in transmission of nerve signals) and other chemicals liberated by dead cells. All these changes have a deleterious effect on the adjoining brain tissue and blood vessels. Diagnosis of stroke syndromes is not difficult in clinical practice. The clinical profile is very suggestive of stroke. For example, the neurological dysfunction occurs with sudden or rapid onset, reaches a maximum in a few hours or days and then the clinical condition stabilises if the patient survives, followed by a fair degree of recovery. Occasionally this clinical profile can be seen in other pathological conditions. Therefore, investigations are necessary, not only to differentiate from other conditions but to ascertain the site and size of infarction. Haematological and metabolic laboratory investigations are necessary for assessment. CT, MRI, MR, angiography, DSA, Doppler studies, SPECT are necessary, but choice of investigation depends on several considerations. The treating or consulting doctor is often asked by the family members, after initial examination, to give the prognosis of the survival of the patient. There is poor correlation between the outcome and investigations in general. CT or MRI at the most can define precisely the structures and the extent of involvement. The

level of consciousness and improvement in consciousness subsequently are good predictors of the outcome. Associated other diseases like ischaemic heart disease, hypertension or diabetes add to the bad outcome and are more often associated with sudden deterioration and death in an otherwise stable condition. There is sudden and unexpected tilt in the clinical condition in the first 4 or 5 days after the onset of the illness and it is therefore very risky to give any definite opinion on the prognosis. Once the condition is stabilised and the patient is conscious, the chances are that the patient would survive, but it would still be difficult to predict the degree of disability and response to rehabilitative measures.

Management of Ischaemic Infarction

From the understanding of the basic processes which lead to ischaemia and infarction, the following facts stand out significantly and determine the steps in the management:

- In the first 48 to 72 hours the maintenance of cerebral blood flow is the most important factor.
- For opening up the collateral channels and the anastomotic circulation, a reasonable blood pressure is essential. Low pressure is very harmful and needs to be corrected.
- Giving oxygen to a stroke patient may lead to a reduction of CO_2 level in blood which has a deleterious effect. High CO_2 level helps in widening blood vessels and so helps in maintaining the circulation around the affected area.
- Influence of metabolic factors on infarction starts usually after 72 hours. High blood sugar level, increased acidity and free radicals have a deleterious effect and need to be treated.
- All therapeutic modalities are directed to limit the infarction and to restore the brain function.

Like myocardial infarction, no time should be lost in initiating the therapy in ischaemic infarction. Old 'wait and watch' policy should be discarded and has no place today in the management of stroke. Ideally each patient should be treated in the intensive care or special stroke unit. General care includes ventilation, circulation, skin care, genitourinary care, gastrointestinal care, and avoidance of infection. There is emphasis nowadays on urgent intervention in acute stroke cases, with efforts to treat these patients within the first few hours of the episode. These patients, therefore, should be promptly evaluated and diagnosed, followed by very early therapy. This requires a team approach.

Hypoxic or anoxic ischaemia, also known as hypoxic encephalopathy, is characterised by impaired consciousness. There are no focal or other lateralising signs of cerebral disease. The cerebrospinal fluid is normal. The basic disorder is a lack of oxygen to the brain. It may result from failure of heart function, circulation, or of the lungs. There are many conditions which can involve either of them. More than one factor may be contributory in any case. Severity of brain dysfunction may vary greatly. Some patients recover to near normal while others suffer from progressive neurological disorder.

There are many factors which can modify the effect of hypoxia (an abnormally low level of oxygen in blood). These include age of the patient, temperature, circulation, drugs, metabolic requirement and others. The younger the patient and the lower the temperature, better are the chances of recovery and survival. A reduction in the metabolic requirement improves chances of tissue recovery. Accordingly the clinical manifestations of hypoxic encephalopathy vary greatly—from mild inattentiveness, poor judgement or motor inco-ordination to coma. Hypoxia beyond 5 minutes usually causes permanent damage, but this time period is not fixed and some persons may survive and recover from 10 minutes of hypoxia or more. In some patients

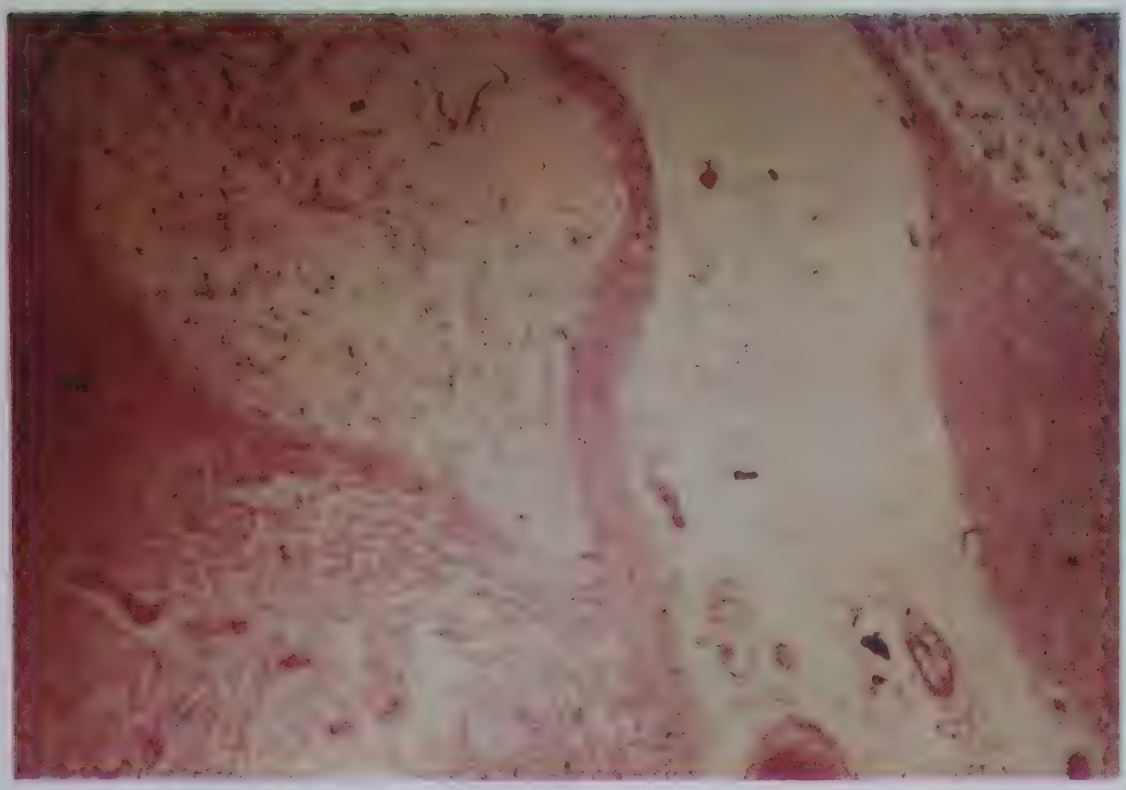

Plate I : Microscopic section of the brain parenchyma showing the chronic changes after infarction. Disruption of tissue and cavitation result.

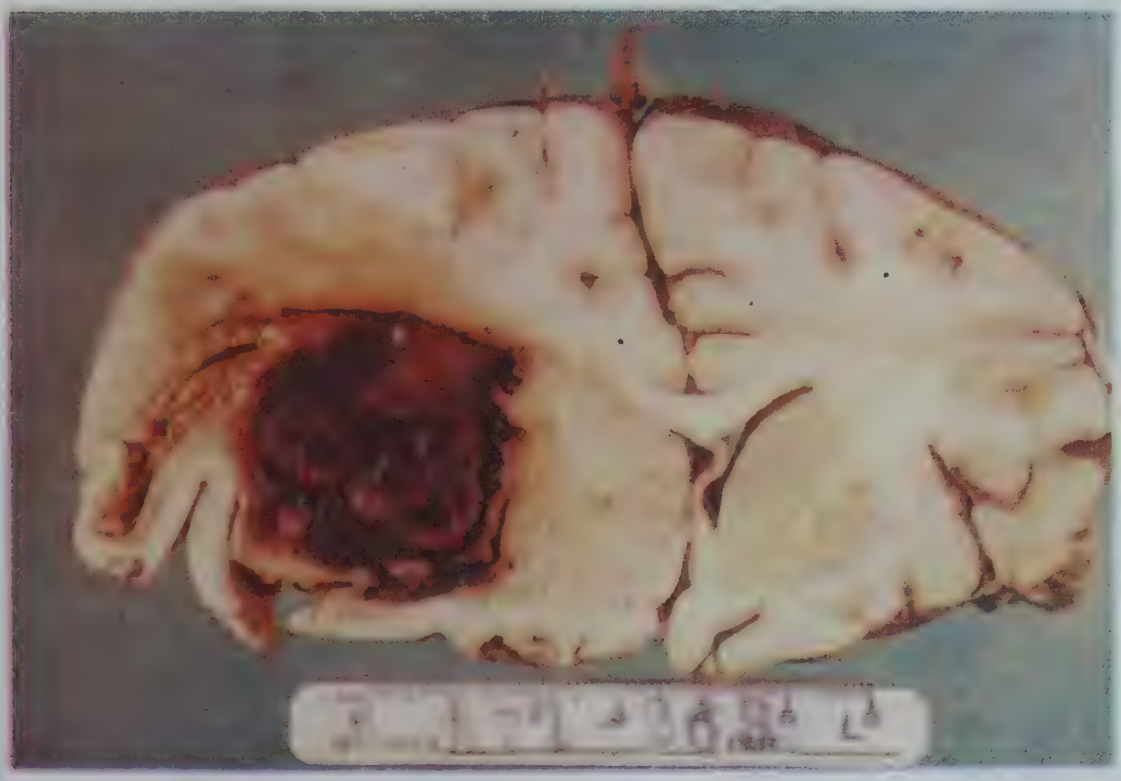

Plate II : Coronal section of the brain showing massive putaminal haemorrhage and surrounding brain oedema.

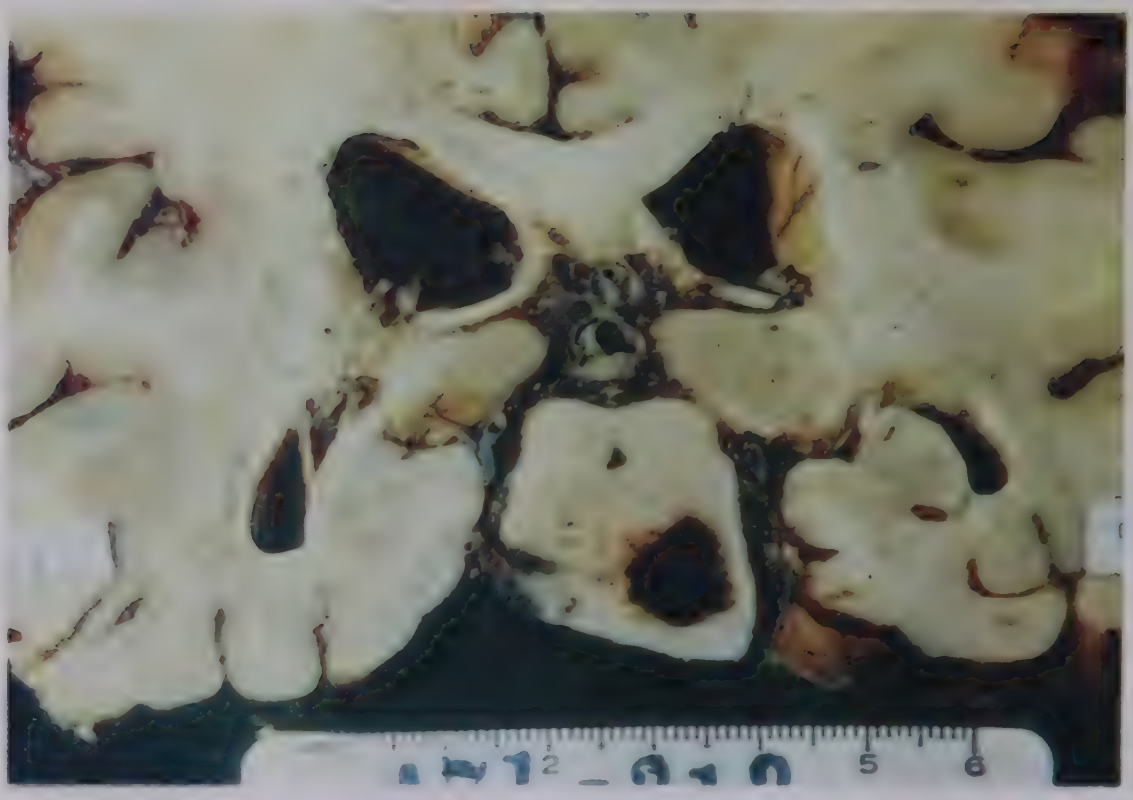

Plate III : Coronal section of the brain showing pontine haemorrhage on the right side.

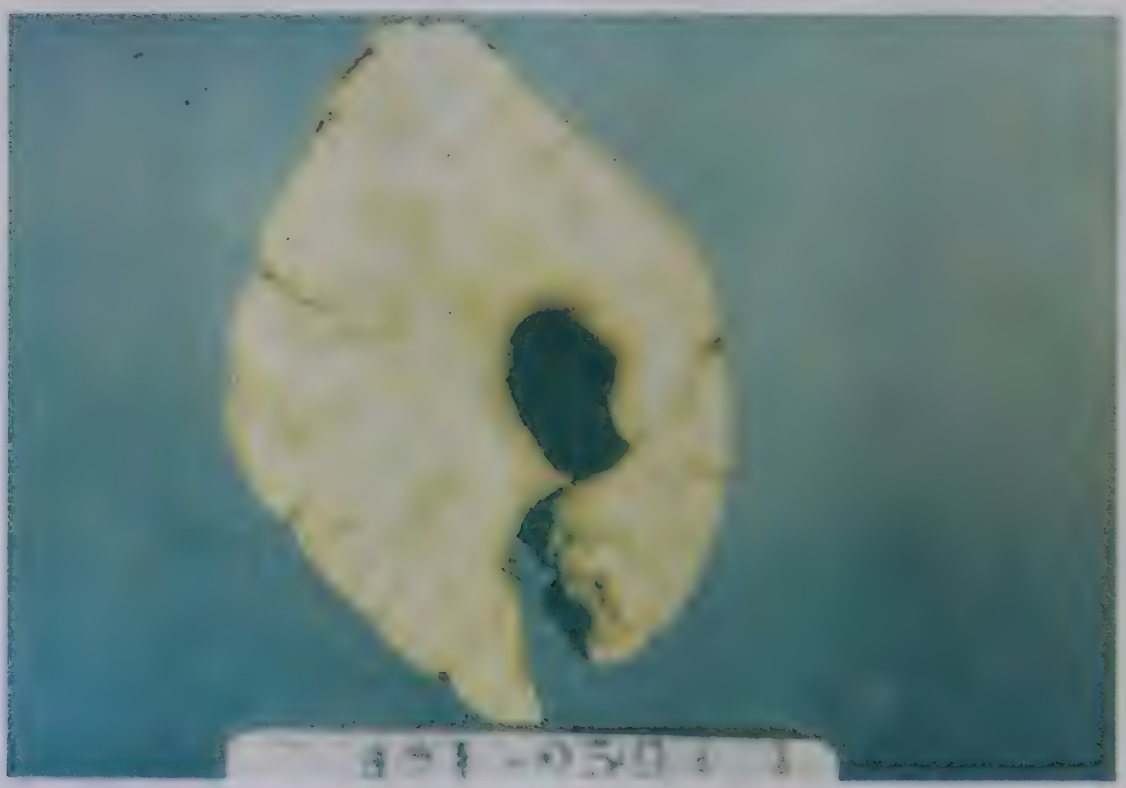

Plate IV : Temporal lobe section showing blood in the temporal horn and destruction of the temporal lobe by haemorrhage.

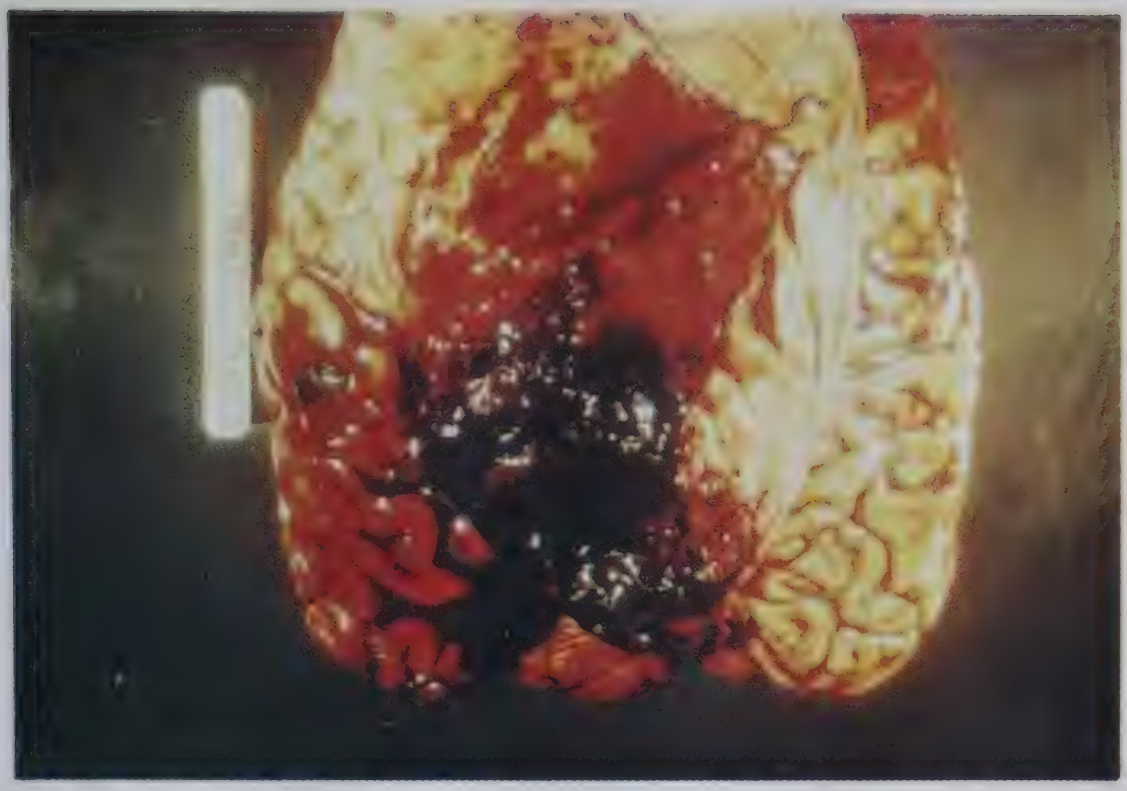

Plate V : Dura mater has been cut to expose the surface of the brain and subdural haematoma (partly clotted).

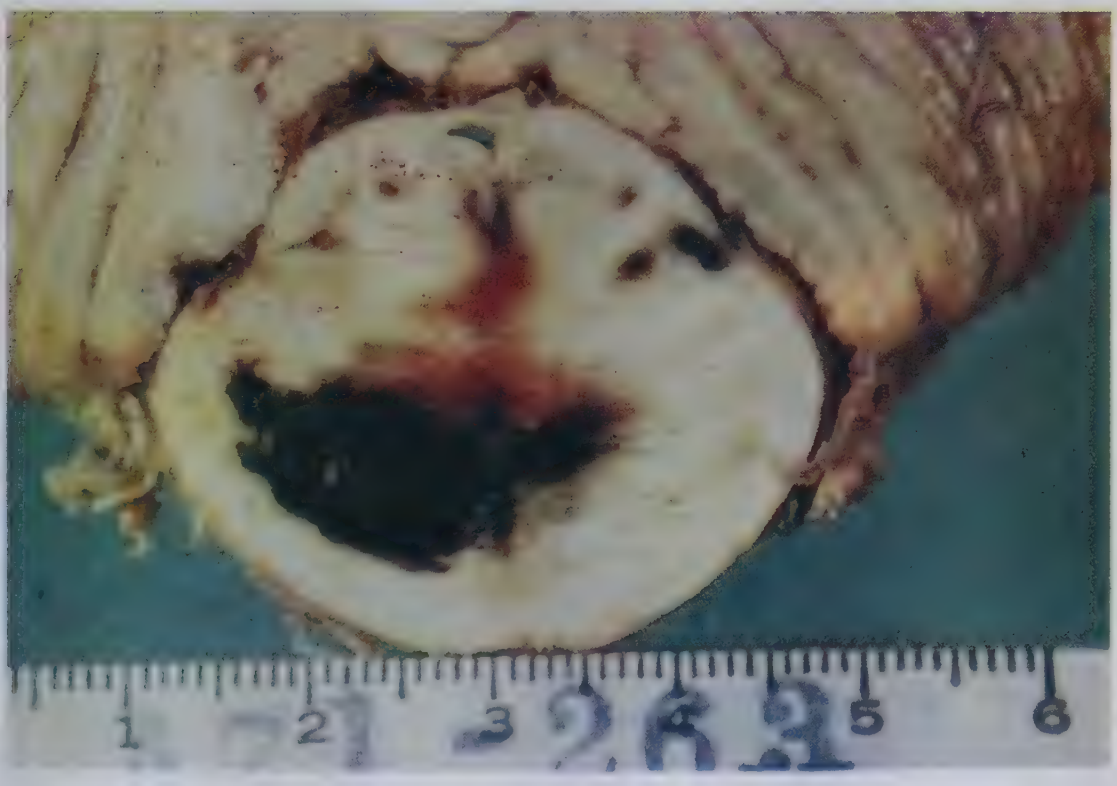

Plate VI : Transverse section of the pons showing primary haemorhage with satellite secondary haemorrhages.

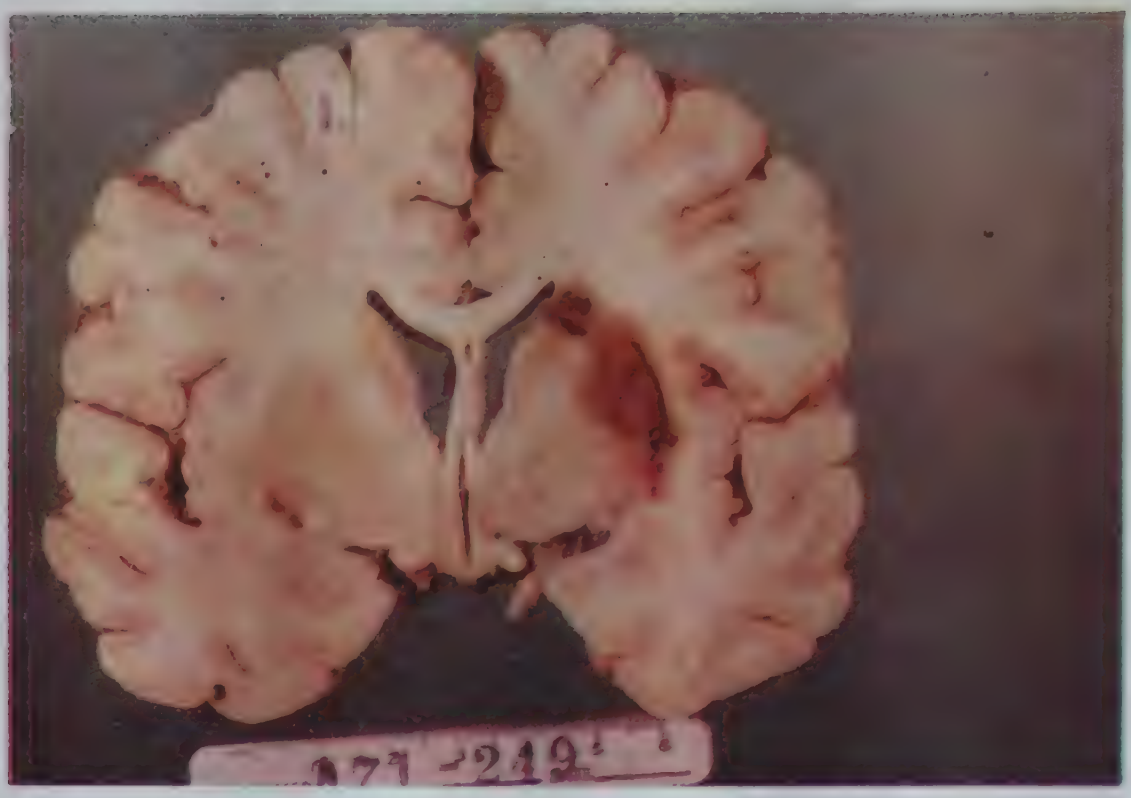

Plate VII : Coronal section of the brain showing haemorrhagic infarction of putamen and caudate nuclei.

Plate VIII : Circle of Willis has been dissected to show the well clipped PCA aneurysm. The patient died of myocardial infarction several years later.

natural respiration cannot be sustained and there are no brain stem reflexes although cardiac action and blood pressure are still maintained. This indicates irreversible damage and is typical of what is known as brain death syndrome. Some patients pass into a state called persistent vegetative state, also known as coma.

Metabolic Infarction

Brain infarction can also be caused by the metabolic constituents and by-products produced in the body. There are many such substances such as glucose, ammonia, bilirubin, urea, alcohol, porphyrins, drugs, and so on. Most frequently encountered metabolic infarction is caused by hypoglycaemia (low glucose level in blood) and is commonly called hypoglycaemic encephalopathy.

Hypoglycaemic encephalopathy is most often encountered in patients of diabetes mellitus on drug therapy, particularly on insulin. Starvation can also be a causative factor. The basic problem is a critical lowering of the blood glucose. The normal blood sugar level is 80 to 100 mg/dl (range varies on methodology and from laboratory to laboratory). If the level falls to 20 mg/dl, the person may develop confusion, but a level below that, about 10 mg/dl, leads to coma. The brain requires glucose for its metabolic activity as a source of energy. There are no large glucose reserves in the brain. The normal brain glucose reserve is 1 to 2 g/100g of brain tissue, and is in the form of glycogen. This glucose reserve will sustain the brain activity for only about 90 minutes. Normally glucose supply is required continuously all the time and is made available to the brain by continuous cerebral circulation. Hypoglycaemic encephalopathy lasting up to 90 minutes usually can be reversed by intravenous administration of glucose, without any neurologic after-effects. Cerebellar cortex is less vulnerable to hypoglycaemia than to hypoxia. General symptoms of hypoglycaemic encephalopathy may be very trivial and

non-specific like nervousness, headache, sweating, palpitation with the patient often gradually drifting to confusion, drowsiness and unconsciousness. Muscular twitchings, and partial or generalised convulsions may also occur. Mild symptoms occur when the blood sugar level is between 30 to 60 mg/dl. A severe and prolonged episode of hypoglycaemia may result in permanent impairment of brain functions. The diagnosis once suspected, is simple and can be established by blood sugar level estimation. Therapy lies in the prevention of hypoglycaemia events and treatment consists of correction of the hypoglycaemic as early as possible. All diabetic patients are advised to have sugar cubes or glucose powder with them and take them at the earliest suspicion of hypoglycaemic symptoms.

Persistent Vegetative State

Some patients at critical and severe stage of illness are saved with the help of intensive care service facilities and other refinements and advances in the treatment now available. They are saved, who would otherwise have died, for an indefinite period, without regaining any meaningful brain functions. Major stroke-ischaemic or haemorrhagic, hypoxic or metabolic encephalopathy, severe brain injury are the usual conditions to give rise to persistent vegetative state. These patients after being in coma for sometime begin to open their eyes, at first in response to painful stimuli and later spontaneously and for increasingly prolonged periods. Intermittently the eyes move from side to side, or fixating momentarily on the doctor or a family member, and giving the erroneous impression of patient's cognition. However, the patient does not speak and shows no signs of awareness of self's inner need or of the environment. The patient's responsiveness is limited to primitive postural and reflex movements of the limbs. These patients can maintain their own respiration without any aid, cardiac function, and circulation. They also have preserved

hypothalamo-pituitary function. This is called persistent vegetative state. Patients in this state can last for months or years depending upon how good nursing care is provided. However, ultimately all die. Such patients really push the health economies in the queer, unmanageable proportion.

9

CEREBRAL HAEMORRHAGE

About 15 to 20 per cent of all strokes occur due to primary intracerebral haemorrhage (brain haemorrhage). They are also called spontaneous hypertensive haemorrhage. Before the CT era all severe and fatal strokes were considered to be due to cerebral haemorrhage. With the routine use of CT and MRI, mild strokes due to intracerebral haemorrhage are recognised which would otherwise have been diagnosed or classified as ischaemic infarctions. With the effective control of hypertension, the incidence of severe and fatal haemorrhage strokes has declined over the years.

There are a number of causes for intracerebral haemorrhage such as trauma, hypertension, bleeding disorders, haemorrhage in tumours, aneurysm and arteriovenous malformations. Primary hypertensive intracerebral haemorrhage constitutes an important separate group in the diagnosis of strokes and requires a different approach for management. The mechanism by which hypertension causes haemorrhage is not very clear in many cases. The common and accepted explanation is the formation of Charcot Bouchard aneurysm (a ballooning of a brain arteriole at the weak point which may rupture). Once the weakened artery or aneurysm ruptures, the blood under arterial pressure escapes into the brain. The escaping blood does not respect any vascular territory and may spread in any direction in the path of the least resistance, and compress and damage the neighbouring structures. The bleeding point is compressed too and causes the bleeding to stop. The severity of clinical manifestations depends primarily

on the size of haemorrhage though the site of haemorrhage is also important. If the haemorrhage is large, midline structures are displaced to the opposite side or downward (Fig.22), leading to coma and death. If the haemorrhage is

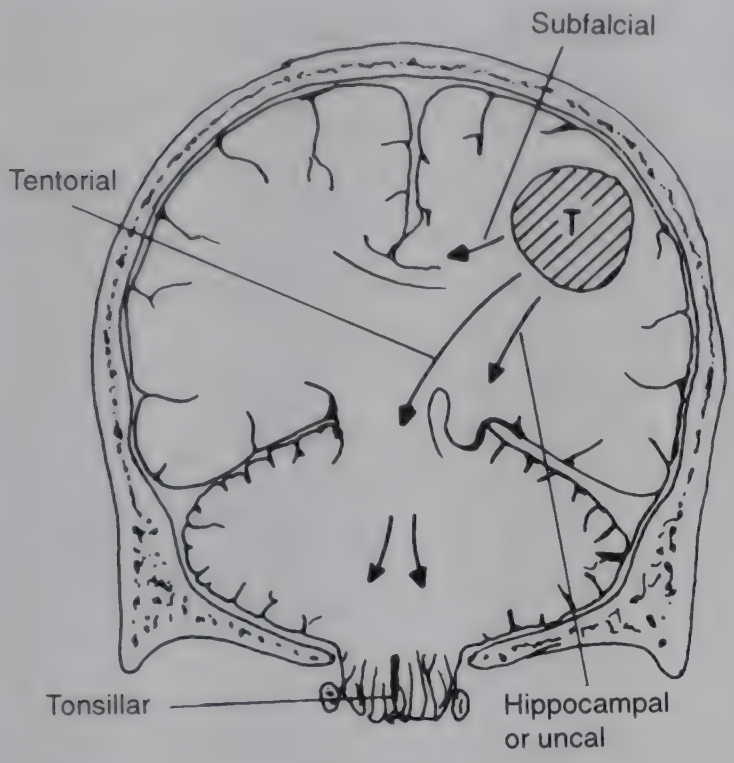

Fig. 22 : The various sites of brain herniation due to mass effect.

near the ventricular surface, the blood can enter the ventricles and circulates along the CSF (Fig.23). It takes a few weeks to a few months for the escaped blood to disappear. Unlike ischaemic strokes, the recurrence of intracerebral haemorrhage is very rare.

It is almost impossible to miss an intracerebral haemorrhage on CT and therefore this investigation occupies the foremost valuable position. Though the diagnosis of intracerebral haemorrhage can be established by proper interpretation of clinical data, CT investigation is necessary to prove the diagnosis, to establish the extent of haemorrhage and associated brain oedema. CT is very useful in making a diagnosis of small haemorrhages which would otherwise be missed. CT investigation is also necessary for differential

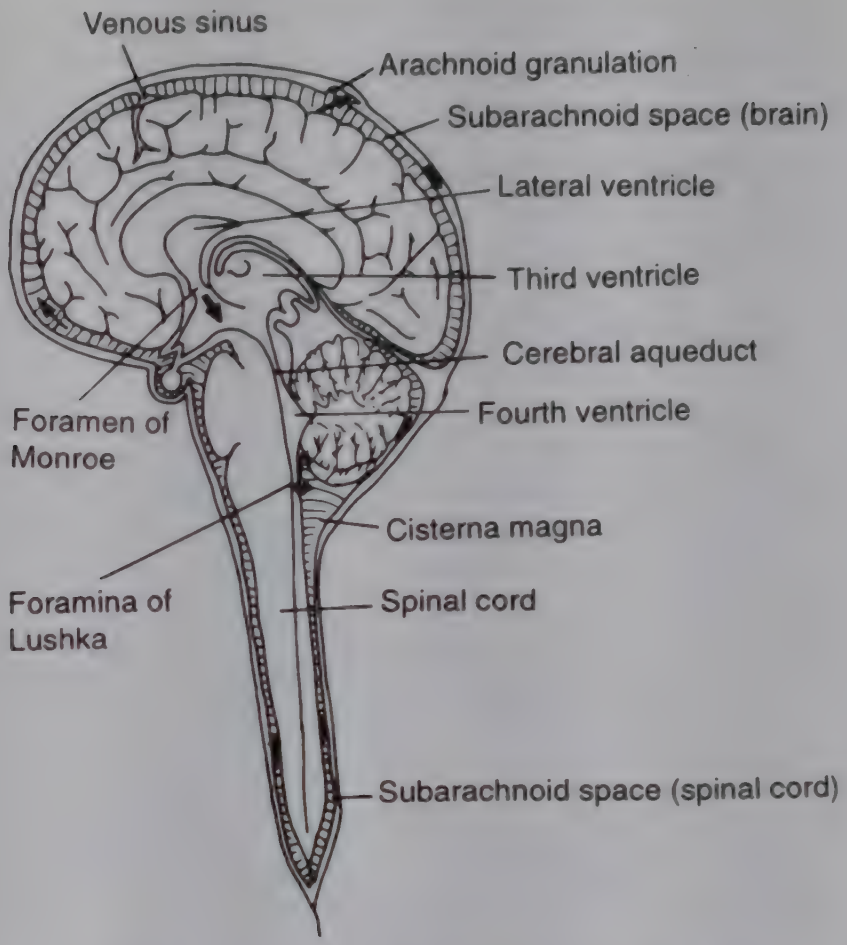

Fig. 23 : The normal circulation of the Cerebrospinal fluid in the brain and over the spinal cord.

diagnosis, exclusion and during follow-up period in the initial stages. MRI is of particular advantage in detecting brain stem haemorrhage. The examination of cerebrospinal fluid (CSF) is not advised these days, as lumbar puncture may alter the CSF dynamics and may even worsen the clinical conditions. Other laboratory investigations may be required to monitor the clinical course and in the treatment of complications.

The best way to control intracerebral haemorrhage of course is to prevent it from happening. The most important preventive measure is the identification of patients at risk and the use of antihypertensive drugs in hypertensive patients. Once bleeding starts in intracerebral haemorrhage, medical treatment cannot stop it. One can only hope that

the bleeding stops on its own. The aim of treatment is to keep the patient alive and get the clinical condition stabilised so that the patient recovers with the least neurological damage. General medical management of all intracerebral haemorrhage is the same as in any comatose patient. This requires care for electrolytes and calories, care of skin, bladder and bowel, respiratory care, avoidance of infection, avoidance of unnecessary physical exertion, and treatment of any complication. It is not advisable to lower blood pressure significantly by drugs as low pressure may even worsen the clinical condition. But severe high pressure needs to be lowered moderately. Use of surgery in the removal of blood, particularly large-sized blood clots, is favoured by some. However, on the whole, surgical removal of blood clot does not alter the clinical outcome very significantly. Once the condition is stabilised, gradual but steady efforts should be made to mobilise the patient. Oral intake and ambulation should be encouraged at the earliest.

Cerebral Oedema

Cerebral or brain oedema is one of the important causes of worsening in ischaemic or haemorrhagic strokes. Brain is placed in the skull cavity which is divided into compartments. This arrangement prevents the brain movement from one compartment to other. In the cranial skull cavity there are two fluid media, blood and cerebrospinal fluid (CSF), which circulate in and over the brain. The volumes of these two liquids are kept constant. Within the skull cavity there is not much available space. If anything from outside occupies any space within this cavity then it is at the cost of compression or displacement of brain tissue, vessels or CSF spaces.

There is also some amount of fluid inside brain cells. This is important for circulation and movement of ions and other constituents and essential for cell life. Brain oedema is

an accumulation of abnormal volumes of fluid in the brain resulting in swelling of cells, tissue or both. Two types of brain oedema have been recognised, vasogenic and cytotoxic. There are two forces working on the fluid while flowing through the vessels. The osmotic pressure keeps the fluid in while hydrostatic pressure pushes the fluid out of the vessel. In vasogenic brain oedema, the wall of blood vessels is damaged, the blood-brain barrier breaks down and the fluid passively accumulates in the extracellular spaces. It is induced by a local increase in the hydrostatic pressure for which there are several causes. Vasogenic oedema is seen more in the white matter of the brain and can be visualised better by CT or MRI. Increased extracellular fluid impairs the function of the tissue and cells.

Cytotoxic brain oedema results from abnormalities in intracellular osmotic pressure. This is best seen in brain injury due to lack of oxygen in which all the cellular elements imbibe fluid from extracellular fluid and swell and results in impairment of cell functions. Cell swelling is correspondingly associated with reduction in the extracellular fluid space.

Brain oedema following ischaemic or haemorrhagic stroke may develop within a few minutes and is worst in first 3 to 4 days. Excessive accumulated fluid is reabsorbed after 4 to 5 days if flow is restored. But if the oedema persists or spreads, brain herniation can result with increasing brain dysfunction and rapid or sudden deterioration in clinical condition, respiratory distress, and death.

Brain oedema requires immediate attention. Efficacy of treatment depends upon several factors but the most important is the underlying condition. Corticosteroids and dehydrating agents are used for controlling the cerebral oedema. Glycerol, mannitol or diuretics are frequently used, but their advantage is limited by the rebound manifestations. Corticosteroids are better in ischaemic or haemorrhagic strokes. Short-term use initiated with intravenous dosage

and then switching on to oral dosage and rapid tapering of the drug in a few days time is probably better and generally accepted. Apart from their effects on reduction of cerebral oedema, the value of these agents in improving the neurological function is very questionable.

10

INTRACRANIAL HAEMORRHAGE

Haemorrhage within the brain cavity can occur in many different sites (Fig.6). There are several different causes that can lead to haemorrhages. The source of bleeding can either be arterial or venous. The basic difference between these two sources is that in the former the bleeding occurs with extreme rapidity and force.

The type called subarachnoid haemorrhage is the third most important and frequent cerebrovascular disorder following atherosclerotic thromboembolism and hypertensive intracerebral haemorrhage.

Subarachnoid haemorrhage may result from bleeding from ruptured artery or vein, or from an intracerebral haemorrhage. There are many causes like trauma, aneurysm, bleeding disorders, hypertension, etc. The commonest cause of subarachnoid haemorrhage is rupture of abnormal balloon-like swelling known as saccular or berry aneurysm. Berry aneurysms are thin-walled pouches that protrude from the arteries in the brain. The size of berry aneurysm varies greatly, ranging in diameter from 8 mm to 2.5 cm.

The common age group affected by berry aneurysm is between 20 to 40 years. Males are affected more often than females. Emotional stress and physical activity have been known to precipitate subarachnoid haemorrhage or are associated with the onset of haemorrhage. A fair number of persons may have trivial or non-specific symptoms before the onset of haemorrhage. A sudden severe headache and neck stiffness are often indications of subarachnoid

haemorrhage. Similarly, sudden collapse after severe headache with relative preservation of consciousness in the beginning is suggestive of an attack. There may be marked clinical variations. About one-third patients may lapse into coma at the onset. There is very high mortality of about 50 per cent. The risk of rebleeding is about 30 per cent during the first week and is greatest between fourth and tenth day. The mortality increases with rebleed.

The diagnosis of subarachnoid haemorrhage is not difficult to make at bedside. CT scan and MRI will easily establish the presence of blood in the subarachnoid space, ventricles or both. CT or MRI may even suggest the site of ruptured aneurysm. However, four-vessels angiography is necessary to demonstrate whether the aneurysm is single or multiple. The neurosurgeon needs to know many things about aneurysm before planning surgery.

Medical treatment consists of general medical care like for any comatose patient. Induced hypotension along with drugs to stop rebleeding is the treatment of choice. Surgical treatment is the best where it can be done safely. Clipping of the aneurysm and wrapping of the aneurysms are the procedures commonly done (Plate VIII). There are other surgical procedures also.

Subdural Haematoma

The space between the inner layer of dura mater and arachnoid mater is called the subdural space (Fig.24). This is present over and around the brain in the cranial cavity and around the spinal cord in the spinal canal. A subdural haematoma is usually the result of an acute haemorrhage caused by rupture of a cortical vein. The most common cause of subdural haemorrhage is injury to the head. Subdural haematoma can be of two types: acute and chronic. Acute subdural haematoma (Fig.19) is usually associated with brain contusion and other evidence of head injury. There may be a time interval between the injury to the head

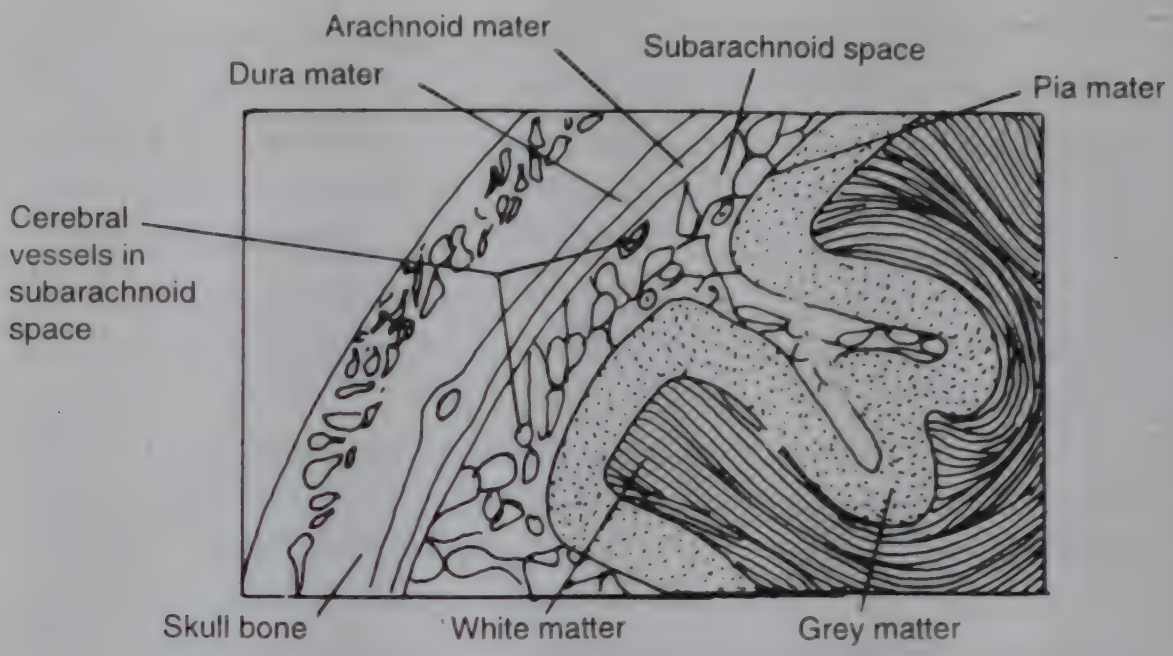

Fig. 24 : Layers on the surface of the brain along with subdural and subarachnoid space.

and the onset of unconsciousness. Other clinical symptoms depend upon the location and compression of the brain and/or vessels. Before the advent of CT, arteriography was routinely done to establish the diagnosis but arteriography is now no longer done. CT is non-invasive and may also reveal underlying skull fracture or brain contusions, etc. There is no other choice than surgical evacuation of the haematoma and therefore the patient's relatives should not waste time in agreeing for surgery irrespective of the patient's condition, age or other factors. Surgery is simple and can be done at the bedside in emergency situation. A dying patient can be revived by this small procedure.

In chronic subdural haematoma on the other hand, the history of head injury may not be available. Injury may be very trivial and most of the time is not told to the doctor at the first instance. Chronic subdural haematoma are frequent in elderly patients and those who are taking anticoagulants or have bleeding disorders. These patients gradually develop drowsiness, confusion, apathy and slowness. The patient may mimic other conditions like loss of memory or even depression. A chronic subdural haematoma can

present like a transient ischaemic attack or simulate a stuttering stroke. Therefore, it is essential to exclude this possibility. Skull X-ray may not be of much value except that sometimes it may reveal an old fracture. EEG may show voltage reduction on the side of haemorrhage. Angiograms will show the displacement of brain away from the skull bone. However, skull X-ray, EEG and angiogram are not required these days. CT without contrast is very useful in the diagnosis and management of chronic subdural haematoma. Surgical evacuation is the best treatment. There are some elderly patients who have no neurological deficit, and on CT done for some other reasons, show chronic subdural haematomas. These persons should be followed up carefully. Surgery may not be required if there is no deterioration in their neurological status.

11

DIAGNOSIS AND INVESTIGATIONS

Diagnosis

It is very essential to make a correct diagnosis of stroke syndromes as the management of the first 4 to 5 days determines the outcome of the patient. The proper and correct diagnosis requires the following steps to be established in the sequential order: (1) Is it a stroke? (2) If it is a stroke then what type is it? (3) What is the site and extent of the lesion? (4) What is the vascular territory of the lesion? (5) What is the extent of functional impairment of the patient? (6) Are there associated diseases? A good working knowledge of the brain and vascular supply is important. The family members and the patient will then be better placed to understand day-to-day events in stroke management.

The diagnosis of a stroke at the bedside is rarely difficult because the mode of presentation is so distinctive. This is called clinical temporal profile. The symptoms develop abruptly or very rapidly, progress in the next few hours or days to a maximum, and then the clinical condition stabilises. If the patient survives then there is fair degree of improvement. Stroke syndromes may be very mild and may consist of trivial neurological signs and may not draw the patient's attention to seek medical advice as happens in transient ischaemic attacks. But it may also manifest with such abruptness that the patient may slip into coma in a few minutes and die as may happen in massive intracerebral haemorrhage or subarachnoid

haemorrhage. There are all grades of severity between these two extremes.

Once the diagnosis of a stroke syndrome is suspected, the neurologist would like to know and establish the type of stroke at the bedside. This seldom presents much difficulty. Some of the important and frequent conditions have been discussed earlier. These have characteristic evolution and temporal profile and pose no great problem. Transient ischaemic attacks, ischaemic thromboembolic strokes, intracerebral haemorrhage, and venous infarcts can be diagnosed. It is the clinical setting in which these stroke syndromes develop that is important for their identification. A stroke following an uncontrolled hypertension is more likely to be caused by intracerebral haemorrhage.

Neurologists would also like to establish the site and extent of the lesion in every stroke syndrome. All functions are located somewhere in the brain at different sites. Anatomical location of these functions is an important prerequisite for establishing the site of lesion. Examination of the cranial nerves is an important exercise as these are distinctly located in the brain. We also know that motor and sensory functions are located on the opposite sides of the brain. Complications of pathological lesions like cerebral oedema, herniation of brain, displacement of brain, compression of vascular channels, etc. may produce their effect and exhibit new signs and symptoms. These would complicate the original symptoms and signs and make diagnosis difficult. Therefore, proper investigation and awareness and knowledge of the events that are taking place are essential for the diagnosis and management of neurovascular syndromes.

Motor weakness, that is, inability to move limbs in one half of the body, may result from several sites within the brain. At one site, a tiny lesion may produce a dense motor weakness while at another site the lesion has to be of large size to produce a similarly dense motor weakness. This has got great bearing on the recovery after a stroke.

Therefore, the treating doctor should try to establish the functional disturbance at a given time in every case.

It is usually important to know and establish the alterations on other organs like heart, lung, stomach or metabolic functions from the brain lesions or stroke syndromes. Some ECG changes are common in stroke syndromes and can be interpreted as ischaemic heart disease. There may be stress ulcers in stomach and can lead to bleeding. A rise in sugar levels in blood and urine is quite frequent in stroke patients and at times may be quite marked in latent diabetic patients and accordingly would require necessary medical management. Electrolyte disturbances, poor intake of fluids may have effect on other systems as well as on the brain. Therefore, laboratory investigations and other diagnostic methods for assessing the systemic diseases or their involvement are important in the total management of the stroke syndromes.

Investigations

The role of correct diagnosis in clinical medicine is indisputable. The correct diagnosis not only saves life or reduces morbidity but greatly influences the health economics. Sequential diagnostic goals have been identified in the management of stroke syndromes in the previous pages. Correct diagnosis requires proper investigations at the right time. There are several investigations available: some are cheap and others are relatively expensive. It is understandable that all investigations cannot be ordered in every case. Choice of investigation and sequence of investigation should best be left to the treating doctor. There are, however, limitations on the choice of investigations in a particular hospital, city or country. Financial constraints also limit the choice of investigations. Therefore, it is very essential for the treating doctor to take all factors into consideration and order the available investigation which can provide the maximum and best information in the management of the

patient. It is a good policy to discuss with the family members or the patient the possible investigation and their role in the management of the case.

There are a number of diagnostic tests. These can be divided broadly into two groups: general and special, i.e. specifically related to stroke syndromes. These investigatory tests can also be divided into (i) non-invasive and (ii) invasive. Computerised tomography has completely revolutionised the diagnostic technologies. Many traditional tests done in the pre-CT era have now been abandoned. General tests may include haematological and biochemical investigations, X-rays, EEG, radioisotope brain scan and cerebro-spinal fluid (CSF) examination, but they will not be discussed here.

Cranial Computerised Tomography (CT)

The different parts of the body like the thorax, abdomen now can be examined by the whole body CT scanner. In stroke syndromes we are interested in cranial computerised tomography (CCT). Different brain tissue slices are examined in sequential order by X-ray beam and with the help of a computer complete skull pictures are developed. These pictures are taken in different directions for better visualisation of different brain regions and structures. CCT can be done with or without contrast media. In stroke syndromes non-enhanced CT is preferred and should always be first done. Blood vessels can be made visible by infusion of contrast media. CT is very good to differentiate between ischaemia or haemorrhage or to eliminate tumours or subdural haematoma simulating acute vascular episode.

Magnetic Resonance Imaging (MRI)

MRI is based on the application of magnetic fields where protons absorb and then re-emit radiowaves. It is possible to change the direction of the magnetic movement of protons

by applying to the sample short bursts of radiowaves of a specific frequency. The radiowaves are analysed by computer and pictures are developed for interpretations.

Advantages of MRI include the absence of X-ray exposure, improved contrast between structures and absence of bone artefacts. In many situations MRI is better than CT, particularly in evaluation of brain-stem lesions. MRI gives better anatomical correlation but it is much more expensive. CT and MRI are complementary tests in the evaluation of stroke syndromes. Both are non-invasive and can easily be repeated during the course of illness.

Recently MRI technology has further advanced. Magnetic resonance angiography (MRA) can partly replace angiography. Without the use of contrast media radiofrequency signals from flowing blood are used to visualise the cerebro-vascular system. Another advance in MR technology is the development of magnetic resonance spectroscopy (MRS). Brain metabolism can be studied by MRS.

Arteriography

The role of arteriography in cerebrovascular diseases has been well established since 1948. It can be used to image the extracranial vasculature in the neck in stroke syndrome, aneurysms, and arteriovenous malformations in the brain, and has played a vital role in the surgical management of strokes. It is equally good to demonstrate subdural haematoma. Major advances have taken place in the development of contrast materials and new imaging techniques, such as four-vessels angiography by femoral route which has provided lots of advantages. The technique has been further developed in order to reduce the complications and take better pictures with the help of the computer. This technique is called digital subtraction angiography (DSA). This can be done by either injecting contrast material into the vein or into the artery. Venous DSA is an almost non-invasive procedure.

Ultrasonography

Improvements in electronics and computer technology have made the utilisation of diagnostic ultrasound for the evaluation of stroke syndromes. This technology is non-invasive, relatively inexpensive and safe. It is of two types: real time (B-mode) imaging and Doppler imaging. B-mode scanning images the arterial wall, not the moving red blood cells. Doppler scanning images the blood flow velocity through vessels. Duplex scan is the combination of high resolution B-mode imaging with Doppler flow detection. It provides the physiologic information of blood flow with 2-dimensional imaging of the walls of the artery or vein. Transcranial Doppler is a new non-invasive technique to measure the flow velocities through the large intracranial arteries.

12

VENOUS STROKE

Any bedside discussion on stroke syndromes gets confined to the arterial system. Venous pathology as a cause of stroke is either not entertained or overlooked, though these have been known to neurologists for centuries. It is true that venous strokes are very rare. But obstruction in venous outflow can cause stagnation of blood and accumulation of injurious metabolic end products, which in turn can cause tissue damage, leading to a venous stroke.

Blockage by thrombus is the most common cause. The thrombosis may be primary or non-infective, or secondary or infective. Dehydration, blood disorders, hormone effect, tumour growth, etc., all can lead to blockage of a vein. Infection of the veins called phlebitis, commonly leads to formation of thrombus and blockage of the vein lumen. Despite the many potential causes, however, disease of the veins is rather unusual. One reason may be that because of valveless flow the blood flows in either direction and allows instantaneous shunting of blood. In clinical practice, infection of paranasal sinuses or middle ear is known to result in intracranial spread of infection.

Clinical features of a venous stroke are very variable and include seizures, weakness, paralysis, raised intracranial pressure, subarachnoid haemorrhage, or coma. The clinical manifestations depend upon the sinus involved, or superficial or cortical veins. Severe headache, vomiting, or seizures in a patient with infection of paranasal sinuses or middle ear should alert the patient and the doctor and should be investigated. The evidence of infection should be

established. CT and MRI are the investigations of choice. Digital subtraction angiography (venous phase) or MRI angiography may show whether the blockage is complete or partial. The use of antibiotics can save many patients. The recovery is rapid and without much residual disability in many patients. Steroids are required at times to reduce the brain oedema. Role of blood anticoagulants and fibronolytic agents in the management of venous stroke is very controversial. Surgical treatment is required if the infection is localised, like brain abscess. Surgical attention will also be required for conditions like severe sinusitis or otitis media, once the brain condition is taken care of. Otherwise, there is the risk of recurrence.

13

STROKE IN THE YOUNG

Stroke as a disease entity is better appreciated in the community by its occurrence in elderly people. Stroke in the young is described as stroke syndromes in persons below the age of 45 years. If one takes all stroke cases in all age groups in the community then it is believed that 15 to 20 per cent cases fall in this group. Stroke in the young causes much more social, economic, and psychological problems. Persons in this age group are mostly bread-winners for the family and the stroke may cripple the family. There are some specific disease conditions leading to strokes which are more common in this age group. For all these reasons stroke in the young is considered separately. One should not forget that primary brain haemorrhage due to hypertension or atherosclerotic ischaemic infarction can also occur within the younger age group.

In general stroke in the young have better prognosis. They tend to make better and more complete recovery. This is probably due to better development of collateral circulation. Prognosis in general is more related to the underlying cause of the stroke. The important factors that may lead to a stroke in the young are disease of heart valves, trauma, dehydration and malnutrition, infection, inflammatory diseases, congenital vascular diseases, blood disorders, migraine, etc.

Strokes in young women related to pregnancy and childbirth are called peripartum strokes. Peripartum strokes are much more common 1 to 4 weeks after childbirth, though they can occur in the second or third trimesters of

pregnancy, and are very rare in the first trimester. Of all strokes in the young, peripartum strokes constitute about 15 to 20 per cent. The risk of peripartum stroke is higher in second or subsequent pregnancies. The risk is more often associated with the poor socio-economic group and home deliveries. The exact causes leading to this condition are still largely unknown, but some factors have been identified which are very often associated with peripartum strokes. These are dehydration after delivery, too much fat intake in the third trimester and following delivery, and alterations in the coagulation profile of blood. Heart diseases, which at times remain undetected, may lead to blockage, particularly valve-related embolism. Peripartum strokes often disrupt the family and cause severe hardship. But they are to some extent preventable by proper antenatal care and hospital deliveries.

Women who take oral contraceptives are at increased risk of developing brain infarction and ischaemic heart disease. Both transient ischaemic attacks and prolonged ischaemic infarctions are known to occur in women who are on oestrogen therapy or on contraceptive pills (which contain both oestrogen and progesteron). The cerebral infarction in these cases is due to blockage of an artery. Hormones make the blood clot easily within the blood vessels which is an important factor in the genesis of contraceptive-associated infarction. The alteration in the cholesterol fractions by progesteron, which increases LDL cholesterol, is another factor. Contraceptive pills should be avoided in those women who have a history of migraine, hypertension and smoking, particularly in child-bearing age.

Inflammation of vessels may lead to blockage of either arteries or veins. There are many other known infectious diseases where stroke-like symptoms may develop during the course of illness. Cerebral malaria is a good example where cerebral symptoms are due to blockages of capillaries.

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VASCULAR DEMENTIA

Dementia is defined as the decline in intellectual functions. Normally, there is some decline in memory and cognitive functions after the age of 40 to 45 years, but it may still be normal for that person and may not interfere with one's functioning in society. To some degree the decline occurs due to loss of brain volume that shrinks during the process of normal aging. Dementia should be differentiated from mental retardation in which there is a failure of development of normal intellectual functions.

There are several causes of dementia which include Alzheimer's disease, brain injury, arteriosclerosis, arteritis, tumours, drug intoxication, degenerative diseases, and endocrine disorders. Until a few years ago dementia in elderly persons was commonly attributed to thickening of arteries, leading to reduced blood flow to brain and consequently, impaired mental functions. Dementia of Alzheimer's type constitutes about 50 to 60 per cent and stroke-related dementia about 20 per cent of all cases of dementia. Recent blood flow and metabolic studies have demonstrated that cerebral blood flow is reduced in both. But persons who develop stroke-related dementia show decline in cerebral blood flow at least 2 years before discernible signs or symptoms occur, while in dementia of Alzheimer's type cerebral flood flow remains normal until dementia is evident.

Stroke-related dementia to some extent is preventable as well as controllable. Control of hypertension and antiplatelet drugs are very important in the management of

stroke-related dementia. The role of drugs, which claim to increase the cerebral blood flow and thereby to improve or halt the dementing process, is very doubtful, but at the same time offers help and satisfaction to the patient. The patient and the family needs a lot of psycho-social support, and for a considerable period of time.

PART II

REHABILITATION

K.K. Singh

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REHABILITATION

A. GENERAL

The purpose of any rehabilitation programme is to provide the patient with the opportunity to reach the maximum level of function and to live the best life he or she can within the limits, if any, imposed by the disease. This is basically a medical programme tailored to meet the needs of the individual in which community reserves may be utilised for the benefit of the patient.

The rehabilitation programme described may be applicable in only a minor degree to any specific patient, or much of it may be found useful. In each case, the programme should be designed to meet the needs of the specific patient and should not be merely the application of a standardised routine.

Another noteworthy aspect is that this chapter deals with patients having 'complete stroke', which is defined as one in which "there is no progression of neurological deficit for 48 hours."

The patient is evaluated serially by a physiatrist, occupational therapist, physical therapist, medical social worker, vocational and psychological counsellor. A comprehensive team approach then places the aims, objectives, goals to be achieved according to the individual requirements of each case.

We hope that the programme described here is sufficiently detailed so that it may be followed where comprehensive rehabilitation services are available, yet sufficiently

simple so that it can be applied when only the services of the family physician and members of the family are available, to aid the patient in the achievement of his rehabilitation goals.

The study can be broadly divided into:

- Evaluation of the patient
- Rehabilitation programme

Before embarking upon the study, a few pertinent questions should be clear in the reader's mind.

Should the patient of 'complete stroke' have rehabilitation?

It has been seen that those who had rehabilitation had better long-term outcomes. Stroke rehabilitation carried out in a rehabilitation centre moves a patient from a low level of functioning to a high level in a relatively short period of time and prevents deterioration of function.

The cost and benefits can be grouped as:

Direct costs

- Hospital bills
- Doctor's bills

Indirect costs

- Modification of home
- Special transportation
- Time lost from employment of family member

Direct benefits

- Improved functions
- Improved performance
- Less dependency
- Employment

Indirect benefits

- Improved quality of life
- Greater socialising
- Greater community involvement

Which patients should be referred to a rehabilitation centre?

All the patients should be referred to a rehabilitation centre in order to have an individually designed rehabilitation programme. After the principal aspects have been taken care of in a rehabilitation centre, some of the long-term aspects can be taken care of at home.

When should a patient be referred to a rehabilitation centre?

Generally some aspects of rehabilitation can be started on the first or second day after the onset of stroke. Certain criteria have to be met, such as having a level of consciousness that permits him to follow two-step, or preferably, three-step directions. Also, the patient should be able to remember and apply today what he has learnt yesterday. If both of these criteria can be fulfilled, then it is not too early to transfer the patient with 'complete stroke' for rehabilitation training.

How soon after the onset of stroke should rehabilitation begin?

Some rehabilitation procedures should begin on the first day, particularly those aimed at preventing the development of complications.

How long does the stroke patient have to remain in a bed or chair?

The stroke patient can start sitting up in bed, or even on a chair beside the bed, as soon as the stroke has stabilised; that is, there is no progression of neurological deficit for 48 hours.

In the past there had been a tendency to leave the stroke patients in bed for too long, for fear of making the

condition worse if the patient started sitting up too soon; but, such a fear is totally unfounded.

Experience with rehabilitation practices applied early to stroke patients have made it apparent that many aspects of stroke that were formerly considered part of the natural history of complete stroke are, in reality, complications that are preventable and avoidable.

Pattern of Recovery in Haemiplegics

Recovery of motor and sensory function is a spontaneous process. The rate of recovery may be influenced by the type of therapy employed but the degree of recovery is probably not influenced by the specific physical therapy employed in the rehabilitation programme.

Maximum return of function occurs within the first three months after the onset of haemiplegia.

The neurological recovery depends upon the mechanism of stroke and localisation of lesion. There is 90 per cent recovery by the end of first three months following onset of stroke except in haemorrhagic types which show neurologic recovery after a longer period.

The functional recovery depends upon motivation to become independent in self-care and mobility, environment and training.

Learning is most satisfactory during the first two-three months after the onset of haemiplegia.

The ability to learn is equal on the paralysed and non-paralysed side.

Final results of rehabilitation are more influenced by the patient's motivation and by social factors than by the degree of physical disability.

To obtain best results in rehabilitation, the programme should be initiated soon after the onset of haemiplegia. This will help prevent avoidable complications.

It is essential to make every effort to motivate the patient to carry out the prescribed programme.

It is also necessary to give consideration to social factors so that on completion of the physical retraining, the patient's rehabilitation within the community is not delayed or brought to a complete halt.

All patients should be initiated into the rehabilitation programme as it is impossible to predict beforehand the progress of the stroke.

Rehabilitation Potentials and Goals

A variety of factors have been found to influence the end result of the rehabilitation programme for haemiplegic patients. Good rehabilitation potential is seen in patients under 55 years of age with left haemiplegia who, show early recovery of motor function and early appearance of finger jerk in the paralytic hand, have minimal or no evidence of mental deterioration, exhibit normal EEG and cerebral angiogram and good motivation.

Poor rehabilitation potential is seen in patients above age 55 with extensive organic brain damage, especially when manifested by psychosis, persistent confusion, poor memory, denial of illness, marked emotional liability and/or apathy, persistent sensory changes, in co-ordination and poor balance, with loss of speech, right haemiplegia, delayed or minimal motor recovery, and changes demonstrable by EEG or vascular occlusion on cerebral angiogram.

Rehabilitation prospects are also reduced by poor pre-illness personality, delay in initiating rehabilitation programme, and rejection of patient by the family.

B. EVALUATION OF THE PATIENT

The evaluation of a disability by a different set of symptoms and signs is important as it differs from the evaluation of a patient for the diagnosis of a disease. Disability diagnosis includes the problem of employment, vocational pursuits,

psychological problems.

The problem-oriented approach can be divided into four phases:

Phase-I	Includes history, physical examination and initial laboratory studies as data base.
Phase-II	Identifies a specific problem list from the data base.
Phase-III	Identifies a specific treatment plan for each of the problems.
Phase-IV	Describes effectiveness of each plan depending upon progress.

History comprises of:

Chief complaint	provides hint of the existing disability.
Present illness	determines the extent of loss of function in basic self-care activities.
Social and vocational history	evaluates environment and provides insight into psychological make-up of the patient.
Review of systems	
Past medical history	

Chief complaint includes statements related to loss of function (e.g. control of leg, (R)-sided clumsiness, trouble with balance, pain in back, leg aggravated by work, etc.). The physician must search for dependency of the patient to carry out his basic self-care skills, problems in social functions in the home and family unit, those related to employment and are secondary to psychological stress.

Present illness elicits the information about the patient's dependency on others which may be standby assistance, or partial or total physical assistance. For example, it is not so much weakness that the patient is concerned about but the reduced ambulation that results. It is not the tremor that worries a patient, but the lost ability to hold the cup of tea.

It is not so much the reduction in shoulder and elbow range of motion that a patient wishes to correct, but rather the lost ability to comb his or her hair or attend to personal hygiene that bothers the patient.

Ambulation includes not only walking but also wheel-chair travel and even, crawling. To assess the extent of disability, the patient's capacity for ambulation in different environments must be obtained.

The environments of significance are his or her home, its immediate vicinity and the community at large. The patient, for example, may be totally independent in walking within his or her house but not in the community or may have reduced community ambulation. If the patient is not walking but uses a wheel-chair, the extent of his or her independence in its use must be known. It should be known if he or she is able to manoeuvre independently within the home or outside the home.

Transfers are movements that involve changes of position in place, e.g. from bed to wheel-chair or regular chair, wheel-chair to toilet/bathroom or coming to a standing position. These activities are more basic than ambulation. For example, while a patient may be independent in walking, he or she may be physically dependent on others to get off his or her chair into a standing position. The independent ambulation is not always available to the patient if the assistance for the transfer into an upright posture is not always present.

Dressing history should be carefully assessed. If a patient is not independent in dressing, he or she is less likely to leave home or receive guests other than his or her immediate family. Dressing dependency therefore greatly restricts the environments available to the patient.

The lost of independence in a patient's ability to feed himself or herself can be most devastating to his or her self-image.

Personal hygiene history includes cleaning, grooming,

tooth brushing, hair combing, shaving, perenial care and the successful handling of bladder and bowel programmes.

Review of systems includes review of the cardiovascular system for symptoms such as dyspnoea, orthopnoea, chest pain, palpitation, cough, etc. It also includes assessment of the respiratory system for cough, sputum, blood in sputum, chest pain and dyspnoea; of the neurologic system for numbness, weakness, fainting or loss of consciousness, dizziness, pain, headache; of the musculo-skeletal system for pain, deformity, weakness, limitation of movement and stiffness. These four systems assess the patient's capacity for specific exercises and training activities.

Past medical history provides information on the residual capacity of patients. A previous disease, trauma or surgery may have produced residual impairments which may compound the disability of the present illness.

Social and vocational history is the source of the necessary data about the environment with which the patient must interact. It identifies the environmental problems and also the personality of the patient regarding his ability to adjust to the stress of chronic disability and thereby the psychological problems that may need to be dealt with, if identified early. This can be split into three types—social, vocational and psychological.

When the patient is dependent on others for basic self-care activities, or a job is lost because of the disease, the identification of secondary and concurrent social issues as patient's problems and inclusion of them in the patient's problem list allows these problems to be dealt along with the pathological process. This includes enquiries about the stability of family unit, its resources, the responsibility of the patient within the unit and the physical environment of the home and the community.

The disability of the patient may also produce employment problem. This problem may then require an

understanding of physical, intellectual and interpersonal requirements of the patient's job. Also, the doctor looks for job instability or adjustment problems.

Vocational activities are also an important aspect of a patient's functions, as some patients derive more pleasure from them than from vocational activities.

Psychological history is important when the reaction of the patient to the stress of the disease is inappropriate or insufficient and when new learning is not occurring during treatment.

Current response of the patient to disease stress is assessed by observing if the patient is regular in his check-ups, taking his medicine, altering his habits, diet, monitoring and prevent secondary complications. This exhibits satisfactory adjustment and the patient is more likely to get over the disability and achieve independence.

The patient can be given certain goals which act as motivational factors towards which the patient works as he strives to remove dependency during treatment.

C. REHABILITATION PROGRAMME

Once the evaluation of the patient is complete, it is proper for the physician to discuss with the patient and his family the nature of the illness, the likelihood of functional recovery, and in general terms, the programme which is to be followed.

For the sake of convenience the programme can be grouped as medical and neurological; physical; psychological and social.

It must however be realised that these proceed generally simultaneously.

Medical and Neurological

These therapies are the elements in any rehabilitation

programme for patients with partial paralysis. The therapies carried out, whether essentially medical or surgical, will depend on the diagnosis, the location of the neurological and vascular lesions, the extent of the damage and the prognosis. This phase of the programme includes not only drugs and specific surgical procedures but also good nursing care and proper attention to such fundamental problems as the patient's cardiac and skeleto-motor functions, respiration, bladder and bowel functions.

Bladder Incontinence (leakage)

After the onset of stroke, the urinary incontinence may be a result of confusion or communication disorder or presence of a flaccid, distended bladder with dribbling overflow type of incontinence. Here catheter drainage may help. Later, as peripheral reflexes return, the bladder function returns to normal.

Urinary sepsis (infection) is likely to occur if the urinary catheter is left for too long. The longer the catheter is in the body, the greater the likelihood of urinary sepsis.

Bowel Complications

Even with severe brain damage, intestinal peristalsis continues so that the faecal matter arrives at the rectum involuntarily. If the patient is unable to inhibit the urge, faecal incontinence results. This especially occurs in conjunction with urinary incontinence and especially if brain damage is more extensive and bilateral.

A good bowel programme should be initiated which includes:

- Encouraging bowel evacuation at a specific time
- Use of laxatives to keep stools soft
- Use of plenty of fluids and roughage in diet

Positioning of Patient

Arrange the cot in the room in such a fashion that maximum activities take place on the affected side. The bed should be flat.

In bed (lying supine)—Keep the affected arm away from the body part (abducted and externally rotated at the shoulder), wrist extended, fingers straight. To facilitate this, place a pillow under axilla and forearm. Initially the patient cannot tolerate this position for long but gradually elevate the hand, which increases external rotation at the shoulder joint.

Paretic leg—To prevent rotation of thigh, place sand-bags at the outer side of the thigh, a small roll under the knee to prevent hyperextension of knee. To counteract tibial rotation (lower leg), place sand-bags at the outer side of tibia (lower leg). To prevent foot drop, keep the ankle at 90° angle with the help of a footboard or a posterior leg splint.

Lying on haemiplegic side

Trunk is slightly backwards, stabilised by pillow at the back and bottom.

Haemiplegic arm lies extended (90° flexion at shoulder, elbow straight, palm upwards).

Haemiplegic leg lies extended, knee slightly bent.

Sound arm rests on the trunk or on a pillow.

Sound leg is kept stepped up on a pillow with the knee slightly bent.

Lying on non-affected side

Trunk is slightly leaning forward.

Haemiplegic arm lies extended on a pillow.

Haemiplegic leg has the knee and hip slightly bent and leg and foot stabilised on a pillow.

Sound arm is kept in a position comfortable for the patient.

Sound leg is extended.

After the initial phase of the illness is over and the

patient is ambulatory, a pillow should never be placed under the paralytic leg to prevent abnormal bending of knee. The positioning should be interrupted by passive exercises as often as possible.

Use of Splints

To prevent severe abnormal bending of the fingers and wrist in patients with marked spasticity, splints can be given to produce extension of the wrist and fingers. The splints in use are the cock-up splint where the wrist is kept in 30° flexion, the knuckle joints in 45° flexion and fingers or interphalangeal joints at 90° pan-cake splint.

Range of Motion Maintenance

After the first day or two of illness, the joints of the affected extremities should be carried through the full range of motion passively. The procedure should be repeated one to three times daily.

When the cooperation of the patient can be enlisted, he should be instructed in a series of exercises that will help to maintain the joint's range of motion. With some patients these exercises can be started a few days after the onset of illness but with others, weeks or months may pass before active cooperation can be enlisted. These exercises should be done slowly so that the movement does not cause rebound stiffness, i.e. excite overactive stretch reflexes in the muscles to be stretched. These should be carried out by the patient to show the physician that he is proficient in his performance.

Exercises

These should be initiated as soon as voluntary movement begins to manifest. The purpose of muscle re-education is

to facilitate, if possible, the return of motor power in the paralysed extremities.

The first voluntary movements that are usually observed are hip and knee flexion, followed by abduction and flexion at the shoulder.

Usually it is possible to begin specific muscle re-education four to ten days after the onset of stroke.

Balance

An initiation of sitting balance is to be done at the earliest. Initially the patient sits on the bed with assistance and one or two days later he should be allowed to swing his legs over the edge of the bed. He could be made to sit with a person standing close by on his paralysed side.

After bed-sitting is mastered, the patient learns to sit on a chair. The chair can be made to have a relatively high seat with pillows placed on the chair.

There are four major factors which account for the difficulties in the balance of elderly haemiplegics. These are:

- Disturbance in cerebral circulation
- Impairment of writing reflexes
- Changes in motor function
- Sensory involvement

Disturbances in circulation with episodes of transient cerebral ischaemia are particularly likely to occur when the elderly patient changes rapidly from a lying to the erect position. To minimise this, it is advisable to make the patient rise slowly from the lying to sitting position.

In the elderly haemiplegics it has been observed that because of impairment of many postural reflexes, quick turning of the head to the left or right or extension of the neck when looking or reaching for an object overhead may result in a loss of balance. So the patient should be advised against rapid head turning and not to reach for overhead objects.

Neuro-muscular Facilitation Techniques

A number of muscle re-education programmes based on neuro-muscular facilitation have been developed and widely used. The widely accepted ones are of four types:

- Bobath
- Brunnstorm
- PNF
- Rood

Bobath is a neuro-developmental treatment programme developed initially for cerebral palsied children and then later applied to stroke patients. The basis of this treatment programme is that a "normal posture reflex mechanism is the basis for normal voluntary skilled movements." A disturbed normal posture reflex leads to disturbed motor performance in adult haemiplegics. The general aim of the exercise is to reduce or increase or stabilise postural tone (depending upon its effect on movement). So, the patient is evaluated for postural reactions, the ability to perform voluntary movements and the state of balance and other automatic protective reactions. The technique of the therapy depends upon the stage of recovery of patient by altering the postural tone, inhibiting interfering patterns or movement reactions and facilitating patterns leading to selective motor control and functional skills.

Brunnstorm is a technique of muscle re-education utilising reflex training for haemiplegic patients. It is based on the basic flexion and extension synergies of the upper and lower extremities along with the associated reactions of haemiplegia evoked by yawning, sneezing and coughing. The associated reactions of haemiplegia are the involuntary limb movements and the reflex tensing.

The technique consists of using the associated reactions of haemiplegia, the synergistic patterns and the pathological tonic neck and labyrinthine reflexes. Resistance to

the normal limb sensory input by tapping and the relaxation techniques are also used. The actual techniques depend upon the stage of motor recovery in the individual patient.

The goal of the Brunnstorm technique is selective motor control and then utilisation of more advanced orthodox muscle re-education techniques.

Proprioceptive Neuromuscular Facilitation (PNF) utilises basic neuro-physiological mechanisms to increase central nervous system excitation and hence, movement. These include maximum resistance, traction and approximation of joint structures, quick stretch, cutaneous pressure with holds and grips, re-inforcement of weak movements with synergistic stronger movements, use of simple verbal commands and basic spiral diagonal patterns of movement.

Rood Technique stresses the concept that sensory input determines motor input. It uses pressure techniques of tapping, pressure and joint compression along with a sequence of development activity programmes.

Traditional Exercise Programming

This exercise programme is comparable to the neuromuscular facilitation programme. For example, an upper extremity exercise programme consists of instructions on range of motion, self-care and activities of daily living along with the use of static splints, dynamic splints and other ortholic devices considered appropriate. Obtaining and maintaining a functional pain-free range of motion in the upper extremities is an important goal.

The programme consists of:

- Choice of proper bed
- Bed positioning, to help prevent contractures
- Footboard, to prevent tightness of heel cord
- Intermittent prone position, to prevent hip and knee contracture

- ROM exercise daily (passive or active, assisted or active)
- Splints or braces
- Tilt table
- Ambulation

If contractures develop, then the following steps should be taken :

- Heat/ice to relax muscle and relieve spasm.
- Stretching (prolonged and gentle; not brief)
- Positioning
- Active exercises
- Ultrasound for moderately severe contracture

EMG Biofeedback

Electrodiagnostic techniques are dependent on the activation and display of electrical activity of the motor unit. This is the physiologic unit of the nervous system. Once the voluntary activity is seen to be returning, a biofeedback plan can be executed when the patient appreciates his muscle power on the screen and can be encouraged.

D. AMBULATION (WALKING)

Ambulation training is begun soon after the patient is able to stand unsupported at a rail. Early in the training, emphasis should be placed on the development of reciprocal gait pattern which characterises normal walking. This is best accomplished if the patient is trained to walk between parallel bars.

At times, due to persistent muscular weakness or spasticity, the patient may adopt a gait which is not of normal character. Under these circumstances, the patient should be taught the gait which is safest for him and which he can use without assistance rather than the gait which has the most normal appearance.

A haemiplegic may adopt any of the following patterns of gait:

Circumduction Gait

This is due to relative lengthening of the leg caused by partial or complete foot drop (extension), stiff knee and equinus foot (reflex plantar flexion, often with supination) during the swing phase with the affected leg. Because of the difficulty involved in bringing the leg forward it is circumducted.

External Rotation Gait

In this manoeuvre, the patient rotates the affected leg externally so that the foot is directly sideways. The patient walks by stepping forward with the good leg and swinging or dragging the externally rotated or paretic leg up, parallel to the good leg.

When the involved leg is externally rotated, it causes the sole to pronate or face enternally, which also helps to overcome the spastic supination or internal facing of the sole of the foot.

Intermittent Double Step Gait

Here the patient takes one step with the involved leg, another with the good leg and stops. The patient appears to lose his balance the moment the weight is placed on the haemiplegic leg, and in order to regain balance, he takes a short step with the good leg and then pauses.

Steps in Gait Training

To learn stance phase the patient stands by supporting himself with a rail. He should take a short step with the unaffected leg forward and back with the affected leg in one

place. This movement is done slowly and frequently till the functional step is achieved.

To learn swing phase the patient steps forward with his affected leg with his unaffected leg stationary. If he is unable to flex his knee with the flexion in the swing phase, he is made to lift his knee and then step with exaggerated knee lift and extension.

Gait drills entail shifting of full weight to the affected leg in a standing position. Gradually if the patient is able to take full weight on the affected leg comfortably, it signifies that he has achieved a standing balance.

Reciprocal gait pattern at the parallel bars is necessary for support during gait training. A four-point cane should be used.

A crutch should not be used ever. A patient who is using a crutch tends to lean away from the affected side by placing weight on the unaffected side. This faulty gait pattern makes progression to cane difficult later on.

Once the patient has developed a good reciprocal gait pattern the four-point cane can be substituted by single-ended cane. After this the patient learns to negotiate ramps and stairs. For this a good mnemonic is "up with the good foot, down with the bad foot."

Use of Braces in Ambulation

In patients with persistent plantar flexion and supination (equinus foot) the brace used should be double-bar, short-leg brace with a 90° stop at ankle. The brace should be attached to an orthopaedic shoe with a lateral 'T' strap at the ankle.

A spring brace in a spastic patient is not used or recommended because the spring may lead to a tendency to ankle clonus (sustained stiffness).

In patients with genu-recurvatum (back knee) the problem is related to hamstring weakness and contractures of the gastrocnemius muscle.

This deformity can be corrected either by progressive resistive exercises for the hamstrings (back of thigh), quadriceps (front of thigh muscles) and gastrocnemius muscle supplemented by correction of contractures and concentration of the patient on a normal gait pattern remedied or by brace-knee cage.

In patients with quadriceps weakness the brace should be double bar with knee-lock on the inner side of leg (to facilitate locking and unlocking).

But this brace may at times be difficult to use as it increases rather than decreasing the patient's handicap.

Patients with rotation weakness or instability at hip can be improved upon by either the elastic spiral strap which is attached at the lateral aspect of shoe and extends across the foot, around the leg, behind the knee and up across the anterior thigh, to a belt at the umbilicus or alternatively by using a long-leg brace with a pelvic band.

To allow greater ease in the swing through the phase of the paretic leg a 12mm or 18mm lift for the heel and a 6mm to 12mm are provided for the sole of the shoe of the unaffected leg.

This simple device would be used as an adjunct to bracing in certain patients, but not as a substitute.

Factors Interfering with Ambulation

Spasticity (resistance to stretching by a muscle) can be reduced but the first question that should be asked is,"How much disabling the spasticity is"?A mild spasticity is welcome as it helps to stabilise the lower limbs and facilitate ambulation.

Only if the spasticity is disabling should it be treated with the help of roll exercises (range of motion), gentle stretching, hydrotherapy, ice therapy, or heat therapy. Drugs like Baclofen, Diazepam, and Dantrolene sodium may also be used.

Certain factors which enhance spasticity should be

avoided. They include pain anywhere in body, contracture (of lower or upper limbs), fear, anxiety, and in co-ordination.

Paralysis and weakness of other muscles play little role in gait interference, except for hip extensors (to be 3/5).

Maintenance of sitting balance can be affected by vision, muscular sensation and proper functioning of the internal ear; two out of these three should be operating. The patient re-learns to develop balance.

Sense of verticality and balance is impaired in some patients but it can be re-learned.

Common Pitfalls in Gait Training

The pitfalls which are commonly met while putting a patient on gait training include inadequate shifting of full weight on to a more involved extremity, which causes development of poor pattern of gait and makes the patient unable to progress in use of the cane.

It is also unwise to wean the patient away from parallel bars till a good reciprocal gait pattern is established or a persistently poor gait pattern exists. Early weaning from four-point cane to single-ended cane is to be avoided. This should not be done until there is no difference in gait pattern whether patient uses a four- or single-ended cane.

E. ACTIVITIES OF DAILY LIVING (ADL)

The activities of daily living encompass:

- Self-care (toilet, eating, dressing and miscellaneous hand activities)
- Home-making
- Walking activities (e.g. using a door, sitting, standing, and travelling activities)
- Non-walking activities (e.g. on bed and wheel-chair)

Self-care Activities

The patient should be instructed to follow the under-mentioned instructions:

- Using his less affected extremity for turning himself in bed. Many haemiplegics tend to lie on the affected side. They can be taught to roll from this position to the supine position and back again, using intact arm and leg. Turning towards the unaffected side is more difficult to master. The patient need not learn to roll over in bed independently before he is started on the sitting balance. The patient should support his affected limbs with the other limbs for turning himself in bed
- Feeding himself
- Observing personal hygiene, such as bathing, shaving, washing, etc.
- Acquiring a sitting position at the edge of the bed without assistance
- Dressing himself
- Transferring himself to a bedside commode or toilet. The toilet seat should be high with a rail used for assistance
- Standing balance
- Going to and from the toilet
- Re-arranging his clothes in a standing position before or after using a toilet

Home-making

This should be started only after the patient attains maximum mobility and communication ability. There are certain pertinent questions that should be clarified before embarking on the home-making programme. These are:

- Whether a patient should work from a wheel-chair or a standing position

- Whether transfer to a stool in a kitchen is practical
- What is the family's general attitude towards the patient's return to home, their expectations of the patient's full or partial resumption of home-making duties
- What is the degree of function in the more involved upper limb which can be used to help or stabilise
- Whether there are perceptual disorders affecting the patient
- Whether speech therapy can be continued at home
- What is the state of the patient's judgement and reliability

The disabled housewife is not the only one who can benefit from training in home-making; men with physical impairment must also assume home-making roles in order to allow other members of the family to work outside the home. Men and women with chronic illnesses are often relegated to a condition of complete dependency. Their potential for independence through training has never been realised. Children and young people with handicaps are certainly candidates for training. Development of home-making skills may contribute to development of self-confidence and initiative.

Home-making activities include care of self, feeding of the family, marketing and shopping, clothing the family, housing and maintenance, family relations, education and recreation, and community participation.

Assistive Devices

Self-help devices or aids for home-making are used for stabilising and holding objects or for extending reach and mobility. In addition, one-handed aids and remote control devices may be useful in patients with limited upper extremity function. Basic considerations in the selection of devices are as follows:

- Use of ordinary tools. Some home-makers hesitate to use tools that are obviously designed for the handicapped
- Home-makers are likely to use assistive devices if allowed a say in their selection
- Consider an assistive device as temporary and change it as the patient acquires new skills

Home-making training involves focus on work simplification in the patient's home, organisation of work and determination of reaching ranges.

Psychological or Psychiatric Help

Perhaps the greatest value in instituting a rehabilitation programme, soon after haemiplegia is its psycho-therapeutic effect; but, the physician must understand and do beyond this to meet the needs of the patient. Family members should bring him hope and optimism rather than depression and despair—a vital factor in motivating him towards recovery.

The patient who has suffered a stroke is brain damaged and for this reason he exhibits certain psychological problems. Because the patient is unable to integrate properly the information he receives from his environment, he makes judgements based on faulty information. This may lead to anxiety, depression, frustration, anger or hostility. In addition the patient is unable to modulate his emotional responses and is likely to express his feelings strongly. He may be quick to anger, stubborn, yet a moment later he may be overly affectionate, laughing and then suddenly crying. It is generally wise to be permissive when the patient is obstinate and later try to gain his cooperation.

The physical and functional improvement which occurs is often very encouraging to the patient. Improvement should be emphasised rather than the deficiencies.

Sensory stimulus from the environment in the form of music, television, news, interaction with family members and relatives is often helpful in checking the intellectual regression.

The emotional liability (loss of control over emotions) is a temporary feature and subsides within a year of the stroke and does not signify weakness of character and attitude.

Easy fatigability occurs especially during various therapies. This should not be confused with lack of interest or depression. Psychological counselling can be resorted to if the patient requires it but only under the direction of a psychiatrist.

Social and Vocational Help

The final social adjustment of the patient depends not only on physical and psychological rehabilitation but also on his family and community. Rehabilitation, after all, must be completed in the home and the community.

Housing is an individual matter and the adjustment needed will depend upon the patient's capacity and on the available facilities.

The outpatient rehabilitation programme continues until the patient has achieved the maximum goal. Vocational rehabilitation is possible in patients who are able to complete a rehabilitation programme. The housewife has the advantage in her job placement as she can work at her own pace, take frequent rest periods, if needed, and complete her tasks with one hand. A great many self-help devices as well as training manuals are available to assist her. Also, she does not encounter the prejudice which is found in industry when a haemiplegic man attempts to find work.

Various levels of work can be considered for the patient, depending upon the degree of recovery and the opportunities available. The patient may return to his job or

to a different full-time job or a part-time job in sheltered workshops for the handicapped, or at home.

The vocational counsellor can be of considerable assistance in evaluation of the patient's work capacity and in actual job placement. Social welfare agencies and welfare organisations can be of help in mitigating the economic handicap.

F. COMMUNITY BASED REHABILITATION

In a community the training programme for a family member or a person who has difficulty in moving, will help the local supervisor to train the person who has difficulty in moving around.

The training programme includes how to move inside the home, around the yard, around the village, and on the roads, and how to travel by bus, boat or an animal, if necessary.

The person to be trained might have been lying down for a long time and may feel dizzy when he or she starts getting up. Teach the person to sit and stand up slowly. This will prevent him from feeling dizzy.

If the person feels dizzy when he or she sits up, help him or her to lie down. After a short rest the person can sit up again but, more slowly than before. Even if the person does not feel dizzy, he or she should stand up very slowly.

To help the person stand up from sitting in a chair, the helper stands on the weaker side of the person. The helper then bends his or her hips and knees to lean forward and holds the person with one arm around his or her back. With the other arm the helper holds the person under his or her bent elbow, forearm, and hand. The person puts his or her feet flat on the ground and leans forward (Fig.25).

The person to be trained may be afraid of falling when first getting up and trying to walk. Stand close by and ready to catch if he or she begins to fall. Let the person know

Fig. 25

that you are there to help. Try to make him or her feel safe. If need be, support the person by standing a little behind and to the side of the person put one arm around the person's chest and with the other arm hold the person's shoulder.

Support the person less and less each time you walk together until he or she is able to walk without help. If the person is afraid of falling, teach the person how to fall.

Put a wide piece of cloth around the person's body, stand behind the person and hold on to the cloth. Ask the person to let himself or herself fall. He should drop the crutches/sticks to the side and fall on to the hands and let the arms bend. Hold on to the cloth, so that he falls slowly; in this way he will not be injured. Repeat till he is not afraid of falling.

If the person is old, do not teach him to fall. Teach the person to walk slowly and carefully, but do not be overprotective.

Small steps should be taken in the beginning as they help to balance more easily. The home should be safe with enough space. If the person needs some support to walk inside the home, teach the person to hold on to the walls, chair or a table for support, or fix a wood or bamboo railing on the walls so that the person can hold these while walking. Keep the things the person needs within easy reach.

The person can be trained to walk by using two bars. Make the parallel bars as shown in Fig. 25, 26. You will need two poles as long as the bars. You can use bamboo or tree branches as poles. These should be at least three fingers thick and strong enough to hold the person's weight (Fig.26.e).

To teach the patient to walk between the bars, ask him or her to stand holding on to the bars, then move one arm

Fig. 26.a

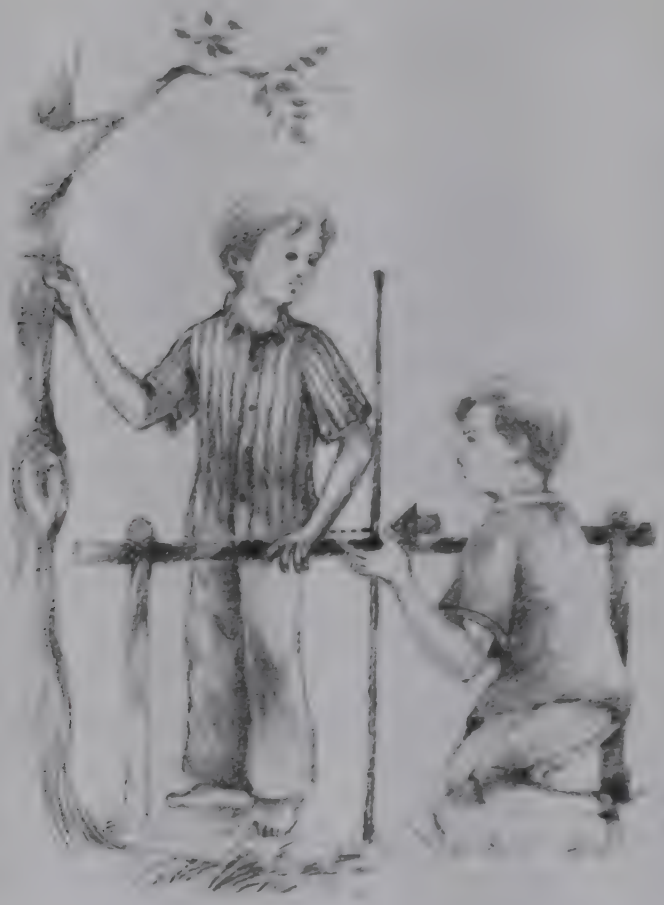

Fig. 26.b

Fig. 26.c

Fig. 26.d

Fig. 26.e

forward on one bar. He or she then moves the other arm forward on the other bar. Next ask him or her to move the weak or painful leg forward. Finally he or she moves the other leg forward. Teach the patient to repeat this, first moving each arm and then each leg (Fig.27).

When the patient first starts walking by using the bars, you may need to support him or her. You can do this by standing behind the person and holding his or her waist (Fig.28).

Fig. 27

Fig. 28

Some patients may need a splint for the knee or the ankle. Consult your local supervisor about this.

Try to let the person walk more and more without your help. Gradually give less support until the person can walk without your help (Fig.29).

If the person you train does not need two bars, train the person to walk holding only one bar. Put the walking

Fig. 29

bar along a path that the person uses. It may be helpful to put it inside the home, in the yard, or along the path to the toilet (Fig.30).

Fig. 30

Teach the patient to walk using one bar by asking him or her to stand holding on to the bar, pushing down with one hand. Then he or she moves the arm that is holding the bar forward. Next, the weak or painful leg is moved forward. Finally, the other leg is moved forward (Fig.31).

Fig. 31

Teach the person to repeat this, first moving forward the arm and then each leg.

If the person needs more support, he or she can lean on a stick held in the hand which is not holding the bar. Or if there is a wall close by, the person can put the free hand on the wall for support.

The person may need a splint for the knee or the ankle. Consult the local supervisor about this.

If the person you train does not need the support of two sticks, you may be able to train the person to walk using just one stick as shown in Fig.32.

To help the patient move the joints explain to the person that it is important to do exercises to move all the joints every day to prevent stiffness.

Help the person daily to move the joints he or she cannot move by himself or herself. Gently move each joint as much as possible in each direction. You can move the joints by doing exercises shown in Fig.33.

Fig. 32.a

Fig. 32.b

Fig. 32.c

Fig. 33

Do the exercises gently. Do not force the movements of the joints when doing the exercise.

Do not cause too much pain to the person.

Sometimes the person may feel a little pain when you begin the exercise. The more the exercise is done the pain would become less.

If the person feels pain when you begin an exercise, and the pain increases with the exercise, stop doing the exercise. Try doing another exercise with the same arm or leg.

If the person feels intense pain with all the exercises that are done for an arm or leg, then stop the exercises. Speak to your local supervisor about it.

For Exercises 9 to 17, the person should be lying on the back. For Exercise 18, the person should turn over and lie on the stomach. If the person cannot turn over alone, help the person to turn over.

Exercise 1. Lift the person's arm up over the head (Fig.33.a). When the arm is beside the head, the elbow can be straight or bent. Then bring the arm down to the side (Fig.33.b). Repeat this exercise six times. Then do the same exercise with the other arm.

Exercise 2. Move the person's arm out to the side (Fig.9.c). Then bring the arm back against the body (Fig.33.d, 33.e). Repeat this exercise six times. Then do the same exercise with the other arm.

Exercise 3. Bend the person's elbow (Fig.34). Then straighten the elbow. Repeat this exercise six times. Then do the same exercise with the other elbow.

Exercise 4. Move the person's forearm so that the palm of the person's hand is turned first towards the person's face. Next turn the palm away from the person's face (Fig. 35). Repeat this exercise six times. Then do the same exercise with the other forearm.

Fig. 34

Fig. 35

Exercise 5. Bend the person's wrist forwards and backwards (Fig. 36). Repeat this exercise six times. Then do the same exercise with the other wrist.

Fig. 36

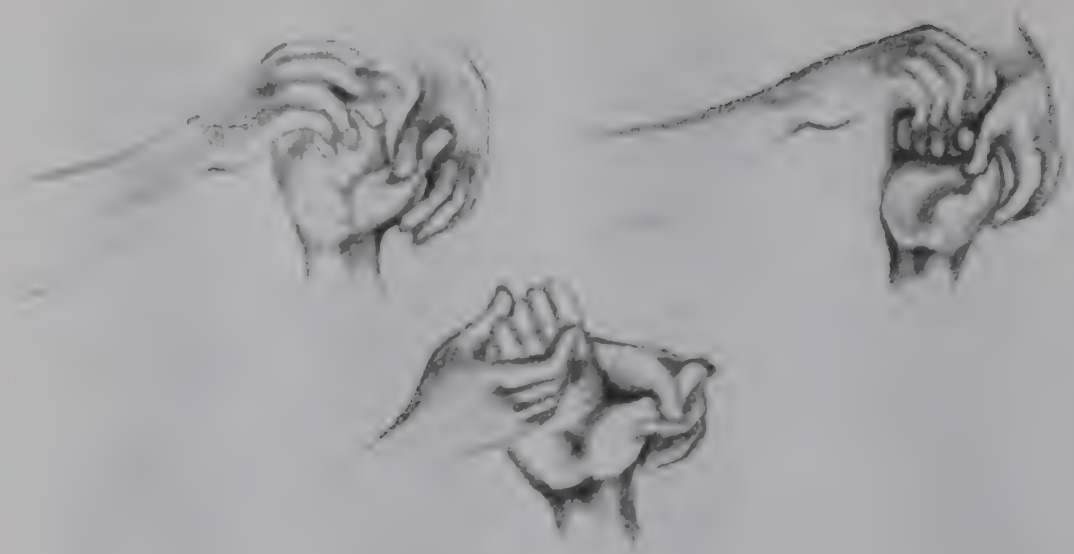

Fig. 37

Exercise 6. Bend the person's fingers and thumb (Fig. 37). Then straighten the fingers and thumb. Repeat this exercise six times. Then do the same exercise with the other fingers and thumb.

Exercise 7. Lift the person's leg with the knee bent (Fig. 38). If the person's other leg does not stay on the bed, put one of your hands on the leg to hold it down. Holding the other leg down also helps to keep that leg straight. Then straighten the leg and put it on the bed.

Fig. 38

Repeat this exercise six times. Then do the same exercise with the other leg.

Exercise 8. Lift the person's leg with the knee straight. If the person's other leg does not stay on the bed, put one of your hands on the leg to hold it down. Or ask another person to hold the leg down. Holding the other down also helps to keep that leg straight (Fig.39). Then bring the leg down on the bed. Repeat this exercise six times. Then do the same exercise with the other leg.

Fig. 39.a

Fig. 39.b

Exercise 9. Move one leg out to the side away from other leg. Do not raise or turn the leg (Fig.40). If the person's other leg moves, put one of your hands on the leg to prevent it from moving. Preventing the other leg from moving also helps that leg. Then bring the leg in again. Repeat this exercise six times. Then do the same exercise with the other leg.

Exercise 10. Hold the person's foot at the back of the

Fig. 40

heel, then pull it down (Fig.41). Then use your arm to move the front of the foot up towards the leg. Next put your hand on top of the person's foot and move the foot down. Repeat this exercise six times. Then do the same exercise with the other foot.

Exercise 11. For the exercise the person should be lying on the stomach. Bend the person's knee (Fig.42). Then straighten the knee. Repeat this exercise six times. Then do the same exercise with the person's other leg.

Fig. 41.a

Fig. 41.b

Fig. 42

To Make a Splint for the Foot

A stroke victim's foot may drag on the ground when he or she walks. Make a splint for the person to wear when walking so that the foot will not drag on the ground.

You will need four straps of canvas or thick cloth three of them three fingers wide and one two fingers wide. The length should be decided depending on where it has to be used (Fig.43.a). You will also need a piece of string or elastic which is the length of the person's leg from the knee to the ground, and a needle and thread.

Put the two wide straps flat on the ground or on a table. The distance between them should be the length of your hand. Fold the narrow strap in two. Fix this strap to the wide straps so that it will lie on each side of the knee, as shown in the drawing (Fig.43.b). Fix the piece of string or elastic to the strap which goes around the foot.

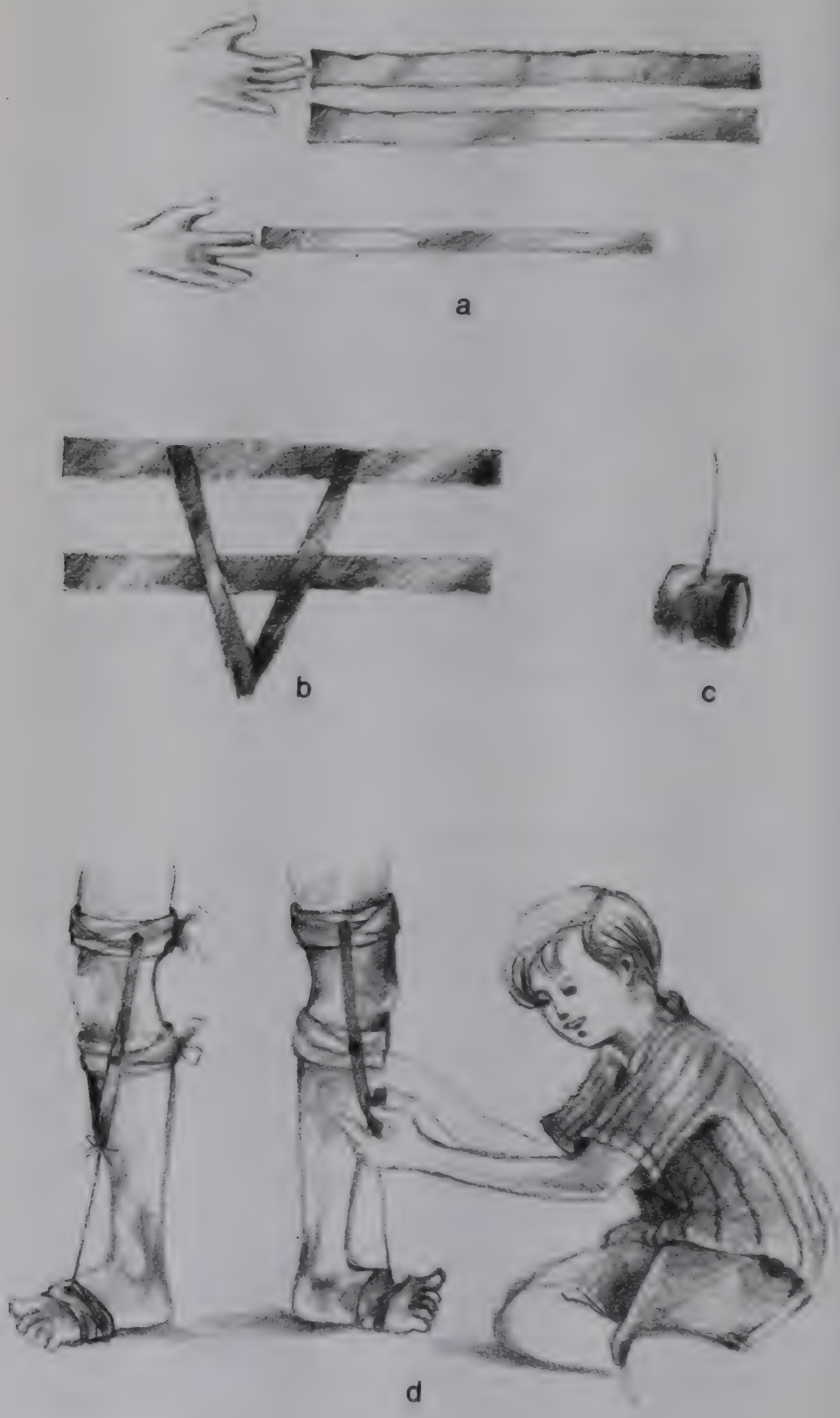

Fig. 43

If the patient does not wear shoes or sandals, make a strap to go around the patient's foot. If he wears shoes or sandals, the strap can go around them.

To wear the splint the two wide straps are tied firmly on the leg. Tie one above the knee and the other below. A narrow piece of cloth holds the two straps together. Put a strap around the foot. The strap has a piece of string or elastic sewn to it. The string should be in the middle of the person's foot (Fig. 43.c). Tie the other end of the elastic to the narrow piece of cloth which holds the straps together.

When the person puts on the splint, tie the string to the narrow strip so that it will hold the person's foot up when he or she walks. Make splints for both feet, if necessary.

Teach the person how to tie on the splint. Put the strap around the foot. Fix the string to the narrow strap (Fig.43.d). Teach the person to take the splints off without help. Walk with the person. Find out if he or she has difficulty using the splint. If the person has difficulty in walking, he or she may need to use sticks. Talk to your local supervisor about it.

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Stroke is one of the commonest neurological emergencies. It is the third most important cause, after cancer and heart attack, of morbidity and mortality in elderly people. Atherosclerosis with thromboembolism is the commonest cause of stroke. Apart from general information, some significant advances made in the understanding of aetiopathogenesis, diagnosis and management of stroke have been discussed in this book.

Dr M.C. Maheshwari teaches at and is chief of the Neurosciences Centre, AIIMS, New Delhi. He has written other very popular books for NBT on most common neurological disorders—*Epilepsy* and *Headache*.

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